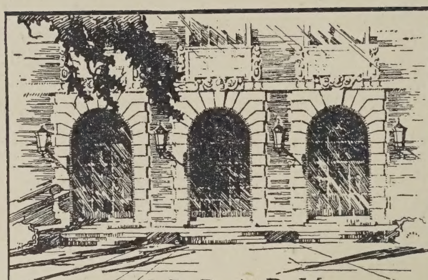

AMERICAN PRACTICE OF SURGERY

A COMPLETE SYSTEM OF THE SCIENCE AND ART OF SURGERY, BY REPRESENTATIVE SURGEONS OF THE UNITED STATES AND CANADA

EDITORS:

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OF NEW YORK CITY

COMPLETE IN EIGHT VOLUMES

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PREFACE.

THE portrayal of surgery as it is practised to-day in the United States and Canada can be best accomplished, we think, by the co-operation of a number of surgeons who have gained eminence in the particular sphere of activity which they are invited to describe. A careful study of the problem which we, as editors, were called upon to solve led us to the conclusion that such a composite work would present a picture more true to life and one more perfect in detail if the different writers were not confined within boundary lines too strictly drawn. While this course would almost necessarily involve a certain amount of repetition, we believed this redundance to be counterbalanced by the fact that the sum total of information supplied could not fail to be greater than if the plan of closer restriction were to be adopted; for experience has shown that no two writers are likely to treat a subject in precisely the same manner or to furnish exactly the same set of details even in important matters.

The first one of the eight volumes of which this treatise is to be composed is herewith submitted to the consideration of the Profession, and we sincerely trust that this product of the combined labor of all concerned will command the respect and confidence of those who consult its pages.

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INTRODUCTION.

THE EVOLUTION OF AMERICAN SURGERY.

By STEPHEN SMITH, M.D., LL.D., New York City.

THE AMERICAN PRACTICE OF SURGERY has had three distinct periods of development, each of which was characterized by conditions sufficiently marked to constitute an era in the history of its evolution.

The first period extended from the settlement of the country to the organization of medical schools—1765–67—and may be called the *primitive* era. During this period there were but few surgeons who had been qualified to practise by a systematic course of education, for to obtain such an education required an attendance upon foreign schools, and few students of that time had the means necessary for such an undertaking. To meet existing conditions the future practitioner was compelled to become an apprentice to a practising physician, and "read medicine and surgery" in his office. His surgical text-book consisted of a copy of Bromfield, Gooch, White, Pott, or any other reputable author of that day, and his diploma, at the expiration of his apprenticeship, was the certificate of his master that he had served the appointed time. There were a few notable instances of surgeons, in the later years of that period, who had graduated from foreign schools, whose practice was of a high order for that time. But their practice was along the lines taught in the schools of London and Edinburgh.

The second period extended from the establishment of medical schools in this country to the introduction of anæsthesia and antiseptics into surgical practice—1846–72—and may be called the *formative* era. During this period the foundations of a distinctly American practice of surgery were laid by the organization of medical schools, in which the future practitioners of surgery in this country were to obtain a competent education.

Two discoveries were made during this period which revolutionized the practice of surgery—anæsthesia and antisepsis. The first abolished pain as a disturbing element during operative procedures, and the second prevented suppuration during the healing process; together they effected a painless operation

and healing of the wound by first intention—results hitherto sought in vain by the elder surgeons. These discoveries swept away the long-established metes and bounds of the field of operative surgery, and made it as limitless as are the diseases and injuries of the human body and man's desire and efforts to relieve them.

The third period, which is now passing, may be called the *practical* era. The surgeons of to-day are making the history of this era, and in this work they will record its marvellous progress for a quarter of a century, and illustrate with historical accuracy the intricate procedures, the instruments of precision, and the vast variety of ingenious apparatus and appliances with which they accomplish results which in the second period would have been regarded as miraculous.

It will be the scope and purpose of this introductory paper to review those conditions of the *formative,* or pioneer, period which gave to the American practice of surgery whatever national traits and peculiarities have characterized its evolution. It will be our aim in this exposition to trace the origin and development of the scientific spirit which inspired and controlled the pioneer surgeons of that period, rather than to record the notable achievements of individuals, with names and dates in due succession, which have only a chronological interest. We shall not attempt, therefore, to follow the precise historical order of subjects, but shall endeavor to secure that continuity of thought, on the part of the student, essential to a just appreciation of the genius of American surgery. The detailed history of American medicine and surgery has been amply written by competent authorities, and can be readily consulted by those seeking specific information as to names, dates, or events.

Two questions arise at the outset of this review which it is important to determine, in order that no injustice may be done to any one who may claim recognition in our narrative, either to the distinction of being a discoverer, or to honors due to a surgeon. These questions are: First, Who is a discoverer? second, Who is a surgeon?

The definition of a discoverer was long since made by the Rev. Sidney Smith, as follows: "That man is not the first discoverer of any art who first says the thing, but he who says it so long, so loud, and so clearly that he compels mankind to hear him." The same opinion is happily expressed by Prof. Howard A. Kelly, of Johns Hopkins Hospital, viz.: "Any claim to priority in medicine and surgery always rests, by consent of the profession, not upon the date of performance, but upon the date of publication." He very pertinently adds: "Reflection will only confirm this dictum by showing that the printed word is, after all, the only possible arbiter which can be appealed to when disputes arise."

The definition of an American surgeon, which will correctly apply to a given specified class of practitioners during the past century and a half, would seem to include the entire profession, for circumstances compelled every physician,

especially during the early part of this period, to perform the duties of a surgeon. But there has always been a class of practitioners who have devoted themselves, by preference, to the practice of surgery and have been recognized as surgeons. The definition of surgeon which is best adapted to the purposes of this work was made by Dr. Valentine Mott, one of the highest authorities on this subject to whom we can refer, who says: "We regard those as surgeons, and those alone, who have, by conscientious devotion to the study of our science and the daily habitual discharge of its multifarious duties, acquired that knowledge which renders the mind of the practitioner serene, his judgment sound, and hands skilful, while it holds out to the patient rational hopes of amended health and prolonged life."

The evolution of American surgery began with the first organized efforts to give the medical students of this country systematic instruction for the purpose of fully qualifying them for practice—1765–67. Whatever other influences may contribute to the formation of the special peculiarities of the practice of a profession, it is the education of its individual members which determines, more largely than any other factor, its individuality. The school formulates the principles which govern the future acts of its pupils. But the quality and value of the instruction of the school depends entirely upon the qualifications of the teachers. Before we can properly estimate the practice of the American surgeon, therefore, we must inquire as to his educational qualifications and then as to the conditions under which he performed his professional duties.

American surgery had its origin in the medical schools of London and Edinburgh. Prior to the organization and establishment of medical colleges in this country, the graduated surgeons took their degrees from the British schools. And during the succeeding half century the more ambitious students of surgery who graduated from the home schools took post-graduate courses of instruction in the schools and hospitals of the mother country. A reference to the teachers of the science and art of surgery in the British schools during this period enables us to form a just estimate of the qualifications of their American graduates to create an adequate system of medical education in this country.

In London we recognize Percival Pott (1713–88), John Hunter (1728–93), Everard Home (1763–1832), John Abernethy (1764–1831), Astley Paston Cooper (1768–1841) as teachers and writers of their times who exerted the greatest power over the progress of scientific surgery. To these more prominent names should be added the names of William Blizard (1743–1835), Henry Cline (1750–1827), Charles Bell (1778–1842), Benjamin Collins Brodie (1783–1862), Benjamin Travers (1783–1858). Of these teachers the one whose genius more completely dominated all others was John Hunter, the most conspicuous figure in the annals of modern surgery. Many American students placed themselves under the immediate instruction of this great master, and their subsequent achievements attest the value of his teachings and example. As surgeon in the

British army in the Spanish Peninsula, 1761–63, he had acquired valuable knowledge of military surgery, which he imparted to his pupils and which they utilized in our colonial wars.

In the Edinburgh school the Monroes—father, son, and grandson—ruled supreme from 1725 to 1846, a period of one hundred and twenty-one years. The elder Monroe was a pupil of Cheselden and was the first professor of anatomy in the University of Edinburgh; he gave clinical lectures on surgery, was a writer of much distinction, and took rank as an authority on many subjects. His son was professor of surgery until 1810, when he was succeeded by his son, who retained the position until 1846. Of the Monroes, the elder was the most eminent as a teacher and surgeon, and by his reputation gave more character to the university school than his son or grandson. For more than a century and a half Edinburgh was regarded as a great centre of medical education, and few American medical students who went abroad to complete their education failed to attend and graduate at the university.

Scarcely less attractive to students who visited Edinburgh during the years 1790 to 1800 was the private school for anatomy, surgery, and obstetrics of John and Charles Bell. John Bell was a bold and fearless surgeon, a brilliant operator, a vigorous writer, and a caustic critic. His school was conducted in opposition to the University Medical College, and the two most prominent professors of the latter—Alexander Monroe and Benjamin Bell—were the subjects of the most unsparing criticism on the part of the founder of the new school. Charles Bell was eminent as an artist, writer, and teacher, and for several years the school was the favorite resort of the more advanced medical students, especially those American visitors who were devoting themselves to surgery.

But few American students, comparatively, visited the schools and hospitals of Paris, except incidentally in their travels. Before 1750 Jean Louis Petit was for a long period the most eminent surgeon of Paris, and it was his genius which gave direction to French surgery. Le Dran and Le Cat (1700–68), contemporaries of Petit, were active in hospital work, and later, in the eighteenth and the beginning of the nineteenth century, the more eminent teachers of surgery in the Paris hospitals were Desault (1730–95), Sabatier (1732–1811), Deschamps (1740–1824), and many others of less note.

Mr. Erichsen, in his address on "Impressions of American Surgery," truthfully remarks: "The method of doing things in surgery is transmitted directly from the master to the pupil; the American surgeon of a past generation acquired in this way the traditionary art of British surgery, and has transmitted it directly to his descendants. Surgeons of both nations drew their inspiration from the same source and drank at the same fountain of knowledge." Though American surgery was originally but a transplanted root of British surgery and its subsequent evolution has been along the inherited lines established by the parent, it is not difficult to distinguish, at many points of contrast, that the

American practice of surgery has always been characterized by a freedom of thought, a promptness of action, and an affluence of resources quite unusual in British practice. Foreign surgeons, accustomed to the observance of technical rules of practice, attributed the independent spirit of American surgeons in their methods of operating to ignorance of the established rules, or to mere recklessness. But experience has proved that the American practice of surgery, from the earliest periods, has illustrated the genius of British surgery, suddenly emancipated from the thraldom with which the traditions of the barber surgeons fettered the progress of scientific surgery in Europe during the eighteenth and the early years of the nineteenth century.

It should be premised that, at the period of the organization of medical schools in this country, surgery had not assumed the position of a science and an art in the medical schools of the capitals of Europe. As a branch of medical instruction it was subordinated to that of medicine, and little else was taught than bandaging and the method of performing the few recognized operations. Whatever didactic surgical teaching was given was in connection with other branches, especially anatomy, and often with midwifery. The more prominent surgeons in the centres of medical education in Europe were struggling to give an independent position to surgery in the curriculum of the schools, but the opposition of the ruling authorities was overpowering during the eighteenth and far into the nineteenth century. And this opposition to a separate chair or professorship of surgery was especially dominant in the Edinburgh school, from which so many American medical students graduated. Educated as were most of the founders of our first medical colleges in the traditions of that school, it is not surprising that, with a single exception, they began to build on similar foundations. That exception was the Medical Department of King's College, New York.

In 1765 the Medical Department of the College of Philadelphia was formally organized, chiefly through the efforts of Dr. John Morgan and Dr. William Shippen, Jr. They were natives of Philadelphia and graduates of literary institutions. They studied medicine in due course in the offices of prominent physicians, the former with Dr. Redman, and the latter with his father, and completed their professional education in the British schools. Dr. Shippen began to give lectures on anatomy in 1762, and annually repeated the course until 1765, when the Medical Department of the College of Philadelphia was organized. In the scheme of instruction the promoters of the school followed the European plan of giving surgery a subordinate place in connection with other branches, and united it with anatomy. Dr. Joseph Carson, historian of the Medical Department of the University of Pennsylvania, says: "The medical school of Philadelphia may be said to be the legitimate offspring of that of Edinburgh."

Dr. Shippen, though not a surgeon, but devoted to the practice of midwifery, was appointed professor of anatomy and surgery, and held this position until

1805, a period of forty years. It appears, from the announcement of his lectures, that the instruction in surgery was limited to "all the necessary operations of surgery" and "a course of bandages." Considering how few operations were regarded as necessary and how important was the use of the bandage, we can estimate the character of the surgical instruction imparted at that time. Dr. Shippen was a popular teacher, and by his devotion to the duties of his professorship, amid the distractions caused by the war of the Revolution, and the dissensions of the profession, powerfully aided in preserving the foundations on which the Medical Department of the University of Pennsylvania, the successor of the College of Philadelphia, had been reared.

In 1767, the Medical Department of King's College, New York, was organized, and on the 2d day of November of that year the introductory address to the first course of lectures was given. In the plan of instruction in surgery adopted by the promoters of this school, we recognize the first departure from European methods and the initial step in creating a class of distinctly American surgeons. The faculty not only exhibited a commendable spirit of independence of the traditions of the past, but they demonstrated unequivocally that they recognized higher ideals of surgery as a science and an art than were prevalent in foreign countries. Under the leadership of Dr. John Jones, surgery was divorced from all other branches of a medical education and erected into an independent professorship. Dr. Jones was appointed full professor of surgery, the first appointment of the kind in this country, and gave the first lecture on the ninth day of November, 1767. He gave an annual course of lectures until the college was closed by the war of the Revolution, 1775.

Fig. 1.—John Jones (1729–1791).

John Jones (1729–91) was of Welsh origin. His grandfather, Dr. Edward Jones, was from Wales, and came to this country in the famous ship *Welcome,* with William Penn and his colony. He married a daughter of Dr. Thomas Wynne, Speaker of the Assembly of Penn's colony. His son, Dr. Evan Jones, settled at Jamaica, Long Island, N. Y., where John Jones was born in 1729. He was educated at a private school in New York, and, at the age of eighteen years, began the study of medicine with Dr. Thomas Cadwalader, of Philadelphia. He visited London and attended the lectures of Dr. William Hunter and the practice of Mr. Percival Pott, in St. Bartholomew's Hospital. In 1757, he again visited France and obtained the degree of Doctor in Medicine from the University of Rheims. In Paris he attended the anatomical lectures of Petit, and received instruction from Le Dran and Le Cat, in Hôtel Dieu.

Dr. Jones began the practice of surgery in the city of New York in 1753, and acquired a wide reputation for skill and success. He performed the first operation

of lithotomy, and subsequently was so successful in this field of practice that he was not only extensively employed in the treatment of calculus of the urinary bladder, but, according to Mease, his success was so great that the operation of lithotomy, which had fallen into disrepute in other States, owing to the failures of operators, was rendered popular.

Dr. Jones enlisted as surgeon in the Continental Army, but soon retired on account of ill-health. In 1780 he removed to Philadelphia, became surgeon to the Pennsylvania Hospital, and was the professional attendant, on occasions, of Washington and Franklin. He died on June 23d, 1791, at the age of sixty-three.

Dr. Jones was eminently qualified to be the founder of a system of surgical education. The qualities of his mind fitted him to be a teacher, and his standard of professional qualification was ideal. He was devoted to surgery as a science and an art, and cultivated it with passionate zeal. He travelled extensively and availed himself of every opportunity to acquire knowledge. Although he made specialties of anatomy and surgery, his general studies took a wide range and his inquiries extended to the collateral sciences. He made warm friends of the most prominent surgeons of that time in the hospitals abroad, and was a favorite student of Pott, of St. Bartholomew's Hospital, London, and of Petit and Le Dran, of Hôtel Dieu, Paris. He attended the lectures of Dr. William Hunter, and must have been brought into more or less intimate association with his brother, John Hunter, who was nearly the age of Dr. Jones and had just completed his studies, 1752. But he probably derived no other benefit from such association than perhaps a more intense devotion to his professional studies.

Dr. Beck, in his "Historical Sketch," remarks of Dr. Jones: "He was well fitted by education and his various accomplishments to become the instructor of others"; and adds: "Not merely as the skilful operator, but as the scientific surgeon and the first teacher of surgery in the colonies, he justly deserves to be styled the *Father of American Surgery*."

Dr. Mease, his student and biographer, thus speaks of Dr. Jones's qualifications as a teacher of surgery: "Viewing the science in an enlarged and honorable light as comprehending the most extensive view of our nature, and as tending to the alleviation and abridgment of human misery, he taught his pupils to despise the servile conduct of those who consider the profession as worthy of cultivation only in proportion to the emoluments which it yields, and to rely upon the solidity of their own endowments as the best security of general esteem and for the acquisition of business."

He taught the twofold nature of surgery—first, as a science; second, as an art—and urged his students to become medical as well as operative surgeons. In the following statement he formulated his opinion of the true surgeon: "Besides a competent acquaintance with the learned languages, which are to lay the foundation of every other acquisition, he must possess an accurate knowledge of the structure of the human body, acquired not only by attending anatomical

lectures, but by frequent dissections of dead bodies with his own hands. This practice cannot be too warmly recommended to the students of surgery. It is from this source, and a knowledge in hydraulics, they must derive any adequate notions of the animal economy or physiology. . . . There must be a happiness, as well as art, to complete the character of the great surgeon. He ought to have firm, steady hands, and be able to use both alike; a strong, clear sight; and, above all, a mind calm and intrepid, yet humane and compassionate, avoiding every appearance of terror and cruelty to his patients, amid the most severe operations."

He made the following distinctions between the qualified and unqualified surgeon: "Whoever has acquired just and general ideas of the nature of a disease will seldom be at a loss how to apply them on particular occasions; and, to him who wants those ideas, no rules or directions will be of much consequence."

He concluded his introductory lecture as follows: "As to those gentlemen who will neither read nor reason, but practise at a venture, and sport with the lives and limbs of their fellow-creatures, I can only, with Dr. Huxham, advise them seriously to peruse the sixth commandment, which is, 'Thou shalt not kill.'"

Immediately on the close of the war the third pioneer medical school was organized. This was the Medical Department of Harvard College, Cambridge, Mass., established in 1782. The plan adopted was that of the foreign schools, anatomy and surgery being united in the same professorship. Dr. John Warren, who was the chief promoter of the school and whose lectures on anatomy before the students of Harvard had attracted much public attention, was appointed the professor of anatomy and surgery.

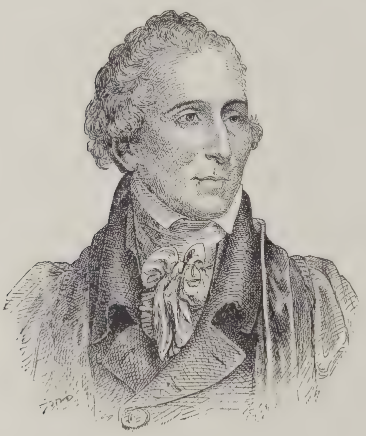

Fig. 2.—John Warren (1753–1815).

John Warren (1753–1815) was born in Roxbury, Mass., on the 27th of July, 1753. He was a younger brother of Gen. Joseph Warren, a surgeon, who fell at the battle of Bunker Hill, June 17th, 1776. He was educated at Harvard College, which he entered at the age of fourteen. He then began the study of medicine with his brother, and on receiving his degree he located in Salem at the age of twenty years. Like his elder brother, Joseph, Dr. John Warren was an ardent patriot, and joined Colonel Pickering's regiment as a volunteer, and marched to the defence of the military stores at Concord. He was present at the first battle at Lexington. He was afterward attached to the main army under the immediate command of General Washington. He was at many important battles, as that on Long Island, at Princeton, and his services were highly appreciated by the Commander-in-Chief. After suffering a severe attack of fever, he was assigned to duty at Boston, where he remained

until the close of the war. In 1780 Dr. Warren gave a course of lectures with dissections at the Military Hospital, and in the following year they were more public and the students of Harvard College were permitted to attend. These lectures led to the establishment of the Medical Department of Harvard, the first course of lectures being given in 1783. Dr. Warren occupied the chair of anatomy and surgery for upward of thirty years. He died April 3d, 1815, of ossification of the valves of the heart and of the aorta, from the symptoms of which he had long suffered.

Dr. Thacher, a pupil of Dr. Warren, thus describes his personal appearance: "He was of about middling stature and well formed; his deportment was agreeable; his manners, formed in a military school and polished by intercourse with the officers of the French army, were those of an accomplished gentleman. An elevated forehead, black eyes, aquiline nose, and hair turned up from his forehead gave him an air of dignity which became a person of his profession and character."

Of Dr. Warren's qualifications to be a pioneer in establishing a system of surgical education, we have ample evidence. Dr. James Jackson, an excellent authority, says: "Dr. Warren's mental attributes were of a high order. . . . His reasoning faculties were acute and powerful. . . . He possessed a peculiar tact for the accurate observation of disease and in rapidly arriving at conclusions. The rapidity of his bodily movements was equally remarkable. . . . His intellectual activity and celerity of motion were manifested in all of the habits of his life."

During the latter years of the eighteenth and the early years of the nineteenth centuries, these pioneer schools underwent many changes in their plans of organization. The Medical Department of the College of Philadelphia became the Medical Department of the University of Pennsylvania in 1791; the Medical Department of King's College became the Medical Department of Columbia College in 1784, and, finally, the College of Physicians and Surgeons in 1810.

But a more important event in the history of these schools than their plans of organization was impending. A new era was dawning which was destined to impart to the instruction in surgery a scientific spirit hitherto unknown. This department was no longer to occupy a subordinate position and be taught in connection with anatomy, chemistry, midwifery, or some other branch, but was to be the subject of an independent professorship, as in the original organization of the Medical Department of King's College, New York.

This change in the system of surgical education was due to the genius of John Hunter, whose researches in the latter half of the eighteenth century gave to surgery the character, dignity, and responsibility of a true science. While the teachings of the British scientist made slow progress in the schools of Europe, on account of national prejudices and jealousies, they early took deep and abid-

ing root in the virgin and fertile soil which the young and plastic medical schools of America afforded, and through which they were to mould the character of its future surgeons. It was a fortunate circumstance that a corps of American students appeared at this critical period in the history of surgery in this country, thoroughly qualified by temperament and education to become the propagators of the principles and practice of the new faith through these pioneer schools.

The first American surgeon, familiar with Hunter's doctrines, who became a professor of surgery in this country was Dr. Wright Post, of New York.

Wright Post (1766–1828) was born at Hempstead, Queens County, N. Y., February 19th, 1766. He was educated by Daniel Bayley, a teacher in that locality. At the age of fifteen he entered the office of Dr. Richard Bayley, a prominent surgeon of New York, as a student of medicine. In 1784, at the age of nineteen, he went to London and became the house pupil of Mr. Sheldon. He became thoroughly familiar with the teachings of Hunter, and was undoubtedly there when that surgeon performed his first operation of ligating the femoral artery for aneurism, 1785. In 1786 he returned to New York and gave lectures on anatomy. In 1792 he was appointed professor of surgery in the Medical Department of Columbia, formerly King's College. From 1796 to 1807, when the College of Physicians and Surgeons was organized, Dr. Post taught anatomy and surgery, apparently without a rival, in Columbia College. He was the first professor of anatomy and surgery in the College of Physicians and Surgeons, but in 1811 the chair was divided, at his special solicitation, and he retained the chair of anatomy, teaching surgery only clinically. In 1813 he received the honorary degree of Doctor in Medicine from the regents of the University of New York, and in 1821 was elected to the office of president of the College of Physicians and Surgeons, a position which he held until 1826, when he resigned. He died in 1828, at the age of sixty-two.

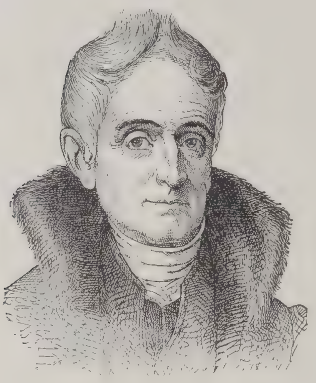
FIG. 3.—Wright Post (1766–1828).

For upward of forty years he was a prominent figure in the medical schools and hospitals of New York, in the former giving didactic and in the latter clinical instruction. In his lectures he taught surgery as a true science, and in his practice he demonstrated it as a high art. He was also the legitimate and worthy successor of Dr. John Jones, being appointed to the chair of surgery in the reorganized Medical Department of Columbia College, formerly King's College, in 1792.

Dr. Valentine Mott, one of his most devoted pupils and eminently qualified to give a judicial opinion, thus characterizes Dr. Post's qualifications as a teacher and practitioner of surgery: "He was unrivalled as an anatomist, a most beautiful dissector, and one of the most luminous and perspicuous teachers I have

ever listened to, either at home or abroad. His manners were grave and dignified; he seldom smiled, and never trifled with the serious and responsible duties in which he was engaged, and which no man ever more solemnly respected. His delivery was precise, slow, and clear—qualities inestimable in a teacher—and peculiarly adapting his instructions to the advancement of the junior portion of the class. As an operator he was careful, slow, and elegant, and competent to any emergency contemplated by the then existing state of surgical science."

In 1811, the College of Physicians and Surgeons, the successor of the Medical Department of Columbia, originally King's College, New York, restored surgery to the position of an independent professorship, which was assigned to it in 1767, and Dr. Valentine Mott was appointed to the chair.

Fig. 4.—Valentine Mott (1785–1865).

Valentine Mott (1785–1865) was born at Glen Cove, Long Island, August 20th, 1785. His father, Dr. Henry Mott, a native of Hempstead, Long Island, was the son of Adam Mott, an Englishman and disciple of George Fox, the founder of the sect of Friends. He was educated at a private school at Newton, where his father practised his profession, and at the age of nineteen entered the office of his relative, Dr. Valentine Seaman, a prominent surgeon of the New York Hospital. Young Mott remained with Dr. Seaman from 1804 until 1807, when he graduated from the Medical Department of Columbia College, in which Dr. Wright Post was the professor of anatomy and surgery. Soon after graduation Dr. Mott visited London, and became the pupil of Sir Astley Cooper, then the foremost surgeon of that city. He speaks of profiting by the teachings of the two Clines, Abernethy, the two Blizards, and Sir Everard Home. He remained in London two years, diligently working in the hospitals and assisting Cooper in his operations. He then visited the Edinburgh school and attended the lectures of Gregory, Home, Duncan, Hope, Monroe, and John Thompson. On returning to New York in the following spring, 1809, Dr. Mott obtained the consent of the trustees of Columbia College to deliver a course of lectures and demonstrations on operative surgery, 1810, in the anatomical rooms of Columbia College. He had to secure his own material by stealth, but he was amply repaid by the success of his lectures, which he claimed were the first "private lectures on any medical subject" in this city, and he states that he was "the first to demonstrate to a class the steps of surgical operations, as then taught and practised by the highest professional authorities." To these lectures he attributed his appointment to the professorship of surgery in Columbia College, 1811, which immediately followed. This appointment was actually made on the advice of his preceptor, Dr. Wright Post, then professor of anatomy and surgery in the College of Physicians and Surgeons, as well as in Columbia College. He continued in this position until 1826, when the professors resigned in a body on account of differences with the regents. The Rutgers Medical College was then organized, and Dr. Mott

entered the faculty as professor of surgery. This school continued but five years. Dr. Mott was next appointed professor of operative surgery and surgical anatomy in the College of Physicians and Surgeons, a position which he resigned in 1834, on account of failing health. He now travelled extensively in Europe, Asia, and Africa. On his return he united in the effort to establish the Medical Department of the University of New York, and in 1840 was appointed the professor of surgery. Though the school had a very able faculty, the fame of Dr. Mott was its greatest attraction to students, and its classes soon far exceeded any hitherto gathered in this city. In 1850 he resigned this position and again visited Europe. On his return he was appointed emeritus professor of operative surgery and surgical anatomy in the College of Physicians and Surgeons, and commenced his course, November 7th, 1850, with an address on "Reminiscences of Medical Teaching and Teachers in New York," an interesting review of the progress of surgery in this city for half a century. In 1852 he accepted the position of emeritus professor of surgery in the Medical Department of the University of New York, which he held until his death, giving annual courses of lectures, chiefly clinical. He died after a short illness from embolism affecting the right leg, April 26th, 1865, his last words being, "Order, truth, punctuality."

Dr. Gross says: "The personal appearance of Dr. Mott was eminently prepossessing. Tall and erect, with broad shoulders and a fine muscular development, he had an open, handsome countenance, a frank, manly expression, and a dignified yet cordial manner. His stature was fully six feet, his forehead high and prominent, the mouth small, the nose aquiline, the chin round and dimpled, the eye large, of hazel hue, and shaded by a heavy brow, and the hair in early life nearly black, with a slight inclination to brownish. His features were regular, and indicative of the benevolence which formed so remarkable a trait in his character."

The late Dr. Joseph M. Smith, who was present as a medical student when Dr. Mott made his first appearance in the lecture-room, says: "When Dr. Mott appeared in the lecture-room of the College of Physicians and Surgeons, soon after his return from Europe, in company with the professor, his appearance made a marked and most favorable impression upon the class. His dress was scrupulously neat, his hair powdered, and his bearing courtly and dignified. All of us regarded him with a feeling of deference amounting to awe."

The qualifications of Dr. Mott as an educator were of the highest order. He was a careful and accurate student of the medical and collateral sciences, and based his practice of surgery upon the principles which they inculcated. He had been trained at home in the school of Hunter by his preceptor, Dr. Wright Post, and abroad by Home, Abernethy, and Cooper. He was the legitimate successor of Dr. John Jones and Dr. Wright Post in laying broad and deep the foundations of surgical education, not only in the schools of New York, but in the colleges of the country organized by graduates of these metropolitan schools.

The successor of Mott in the College of Physicians and Surgeons, New York, was Dr. Alexander H. Stevens, a graduate of the University of Pennsylvania in 1811. Stevens was trained in the school of Physick, and learned the science of

surgery as taught by Hunter. His graduating thesis was "On the Proximate Cause of Inflammation," which involved a discussion of first principles; but so well did he accomplish his task that his essay received complimentary notices from Dr. Rush and others. His teaching was characterized by a thorough exposition of each subject in simple but terse language, with quaint and striking illustrations.

Alexander Hodgden Stevens (1789–1869) was a native of New York City. He was a graduate of Yale College in 1807, and of the Medical Department of the University of Pennsylvania in 1811. In 1814 he was appointed professor of surgery in the New York Medical Institute, and in 1818 he became one of the visiting surgeons to the New York Hospital. In 1826 he was appointed professor of surgery in the College of Physicians and Surgeons, as the successor of Mott. He retired in 1838 on account of failing health, and was made emeritus by the board of regents. He died in 1869.

The true successor of Stevens was Dr. Willard Parker, though the chair of surgery was occupied for two sessions by Dr. Alban G. Smith, of Kentucky. Parker became the professor of surgery in 1840, having been called from the Cincinnati College, Ohio. He was a graduate of the Harvard Medical College and a private pupil of Dr. John C. Warren. Thus it happened that the chair of surgery, once occupied by Jones and Post, then made illustrious by Mott, one of its own pupils, next filled by a representative of Physick, was now to be given to a student of Warren.

Parker had many of the qualifications of the best class of teachers. His very presence and personality commanded the confidence, respect, and even admiration of students. His mental attributes and his temperament rendered his teaching practical rather than theoretical and speculative. He readily grasped the essential facts of any subject matter, and at once endeavored to estimate their practical value. This peculiarity of his teaching was attractive to students and practitioners, and always gave him large and attentive audiences. His special characteristics as a teacher of surgery were seen to the best advantage in the clinics which he organized in the lecture-room of the college, the first of the kind in this country. Here, in a familiar manner, he illustrated the diagnosis and treatment of surgical diseases and applied the principles which he taught to practice. The influence of such instruction, continued for a generation, upon the practice of surgery in this country cannot be estimated.

FIG. 5.—Willard Parker (1800–1884).

It is certain that scores of graduates from the school during that period became reputable practitioners, and many attained to distinction as teachers.

Draper, his student and biographer, states that it was "in his character as a public teacher that Parker impressed himself most powerfully upon all who came within the sphere of his attractions. He loved to teach; he was inspiring and suggestive; there was something about his enthusiasm that was contagious; he never failed to be interesting and to inspire others with something of the energy that swayed his own soul; he was always aspiring to the highest and best in professional knowledge, and was constantly helping to lift others, ambitious to attain it, to a higher plane."

Willard Parker (1800–1884) was born September 2d, 1800, in Hillsboro, N. H. He graduated from Harvard College in 1826, and in 1827 was appointed house physician to the Marine Hospital at Chelsea, where he spent two years. He was a pupil of Dr. John C. Warren, and served one year as house surgeon in the Massachusetts General Hospital. He graduated in medicine from the Harvard School in 1830, and soon after accepted the professorship of anatomy in the Berkshire Medical College at Pittsfield, Mass. In 1832 he delivered a course of lectures on surgery in the same institution. In 1836 he was appointed professor of surgery in the Cincinnati Medical College, and in 1839 accepted the professorship of surgery in the College of Physicians and Surgeons of New York, a position he held thirty years. On retiring he was appointed emeritus professor, and continued in that relation to the college until his death in 1884.

In the year 1805 the teaching of surgery was divorced from anatomy and obstetrics, and erected into an independent professorship, by the governing body of the Medical Department of the University of Pennsylvania, originally the Medical Department of the College of Philadelphia, and Dr. Philip Syng Physick was appointed to the new position. He was admirably adapted by intimate association with Hunter, in his experimental work, to inaugurate in that pioneer school the doctrines taught by his master. It was the concurrent testimony of contemporary writers that Dr. Physick's teaching of surgery placed it on a rational and enduring basis as a science and an art.

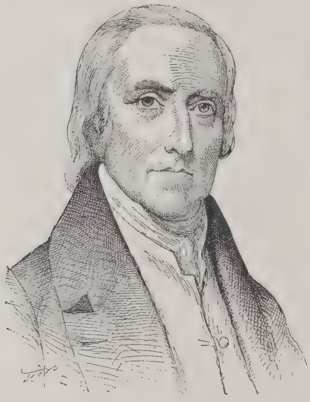

Fig. 6.—Philip Syng Physick (1768–1837).

Philip Syng Physick (1768–1837) was born in Philadelphia on the 7th day of July, 1768. He was of English descent and received his collegiate education at the University of Pennsylvania, from which he graduated at the age of seventeen. He studied medicine in the office of a physician for three years, and in 1789, at the age of twenty-one, went to London and became the private pupil of John Hunter and a member of his family. He was appointed a member of the house staff of St. George's Hospital through the influence of his master, with whom he seems to have been a favorite,

for at the close of his residence Hunter requested him to become his partner. He visited Edinburgh and remained a year attending a course of instruction, receiving his medical degree in 1792, at the age of twenty-four.

Dr. Physick returned to Philadelphia and began practice under the most favorable conditions. In 1794 he was elected a surgeon of the Pennsylvania Hospital, and in 1800 he was invited to lecture on surgery to the students in the university school. In 1805 the professorship of surgery was created in the university, and Dr. Physick was appointed to the chair. He held the position thirteen years, when he relinquished it and accepted the chair of anatomy, which he retained until 1830 in co-operation with a colleague. He died in 1837.

Dr. Physick is described as a man of medium height, with pale, regular, classic features; in manners reserved to the degree of shyness; as to health, the victim of indigestion and catarrh; and in temperament pessimistic, forbidding, and devoid of a sense of humor. In the daily routine of practice he was prompt and precise, requiring his patients only to answer questions, and never allowing them to indulge in explanations. The same precision marked his operations, and, having a small, delicate, and facile hand, every step in the procedure was taken with an accuracy that impressed on the student a most useful lesson.

Dr. John Bell remarks: "Dr. Physick was from this time in possession of the widest field for the exercise of his talents." He was "listened to by the large class in the university, through the members of which he could disseminate the principles of surgery imbibed from his celebrated preceptor, John Hunter—strengthened and enforced by his own meditation and personal experience obtained in hospital and private practice."

Dr. Joseph Carson, the historian of the Medical Department of the University of Pennsylvania, thus speaks of Dr. Physick's method of instruction: "The lectures were carefully written out and delivered with the manuscript before him or in hand; for it was an axiom with him that, on so important an occasion as the instruction of youth in an art so necessary to the well-being and happiness of mankind, every care should be taken to render the inculcation of principles and practice clear to the comprehension of students. . . . His dignified bearing and imposing presence, his emphatic manner, and painstaking execution of his duties deeply impressed his pupils and commanded the profoundest deference."

Intimately associated with Dr. Physick in the duties of his professorship, from the year 1807 to 1818, was his nephew, Dr. John Syng Dorsey.

John Syng Dorsey (1783–1818) was born in Philadelphia, December 23d, 1783. His mother was a sister of Dr. Philip Syng Physick. He was educated at the Friends' School, Philadelphia. At the age of fifteen he entered the office of his uncle, as a student of medicine, and in 1802, at the age of nineteen, he graduated from the Medical Department of the University of Pennsylvania. In the following year, 1803, he visited London for the purpose of continuing his studies, with letters of introduction from his uncle to Sir Everard Home, the brother-in-law of John Hunter, then

one of the most eminent surgeons of that city. He next visited Paris, where he was attracted by Boyer, whom he most frequently mentions in his correspondence. He returned in 1804 and began practice in Philadelphia. Soon after, he became the prosector to his uncle, the professor of surgery in the university, and in 1807 he was appointed adjunct professor of surgery. In 1808 Dr. Dorsey was appointed one of the surgeons of the Pennsylvania Hospital, and in 1813 he published a work entitled "The Elements of Surgery," in two volumes. In 1818 he was appointed professor of anatomy on the death of his predecessor, Dr. Wistar. Dr. Dorsey, however, was not destined to enjoy the advantages and the honors which this position gave him. He delivered a brilliant introductory to the course of lectures which he had planned, on the second day of November, 1818, but was attacked with typhus fever on the evening of that day, and died on the twenty-third day of the same month, at the age of thirty-five.

The appointment of Dorsey to the position of adjunct professor was rendered necessary on account of the frequent attacks of illness of his uncle, thereby rendering his attendance on lectures uncertain. Dorsey was an accomplished lecturer and a practical surgeon of great skill. Professor Chapman, his colleague, says that, with the exception of Dr. Physick, "He was indisputably the most accomplished surgeon in our country, and this high praise is conceded to him on account of the number and variety, the difficulty of his operations, and the skill, dexterity, and boldness with which they were performed. Clear in his views and of sound judgment, he had great mechanical ingenuity, delicacy of touch, and promptness of decision; and hence in conducting an operation, however new or complicated, there was a tone and firmness of manner which always inspired confidence and success. As a teacher of medicine his merits were great and universally acknowledged." He was also an artist of such skill as to attract the favorable notice of persons prominent in that profession.

Dr. William Gibson (1788–1868) was the successor of Physick, and maintained the reputation of the University Medical College, Philadelphia, for thirty-six years. He was a graduate of Princeton College, where he took high rank as a classical scholar. He pursued his medical studies in Edinburgh, and was a pupil of John and Charles Bell. He graduated from the Edinburgh school with distinction, his thesis on "Necrosis," written in Latin, attracting attention on account of its classical style. On his return he was appointed professor of surgery in the University of Maryland, and in 1819 he succeeded Physick in the chair of surgery in the Medical Department of the University of Pennsylvania, a position which he held until 1855. He died in Savannah, Ga., in 1868.

Gibson was a teacher of surgery who always interested students by his positive and earnest manner of delivery and by his accuracy and clearness of statement. In 1824 he published his "Institutes and Practice of Surgery," which passed through several editions and was for many years the accepted text-book of surgery. Thus as a teacher of surgery in the largest medical school in the country

and as author of the text-book of its theory and practice which was widely accepted by the profession, Gibson was one of the early promoters of a sound surgical education for upward of a generation.

Dr. Henry H. Smith (1815–90) succeeded Gibson in 1855. He was a native of Philadelphia, and received both his collegiate and medical education at the University of Pennsylvania, where he graduated in medicine in 1837. He was the first of its graduates to be appointed to its chair of surgery. He is spoken of as "excellent and unexceptional in his style of speaking—quiet, self-possessed, systematic, and thorough." His most important contribution to the practice of surgery was a work entitled "A System of Operative Surgery." He resigned the professorship of surgery in 1871, and was appointed emeritus.


Fig. 7. — D. Hayes Agnew (1818–1892).

Dr. D. Hayes Agnew (1818–92) succeeded Smith in 1871. He was a native of Lancaster, Pa., and a graduate from the University Medical College, Philadelphia. After a brief residence in the country, he located in Philadelphia, and began private teaching of medical students. His success as a teacher was very great, and he attracted large classes of students from all sections of the country. In 1854 he was appointed one of the surgeons of the Pennsylvania Hospital, and while in that position he created a pathological museum. In 1863 he accepted the position of demonstrator of anatomy and lecturer on clinical surgery in the Medical Department of the university. In 1870 he was selected as professor of operative surgery; in 1871 he became professor of the principles and practice of surgery in the same institution.

Agnew fittingly closes the history of surgical teaching in the Medical Department of the University of Pennsylvania during the period under review. Instructed in the policies of this school, and already distinguished as a surgeon and skilled as a teacher by a large experience as an instructor in anatomy, pathology, operative and clinical surgery, he entered upon his duties not only qualified to maintain but to advance its reputation as the true exponent of the science and art of surgery as formulated by Physick.

In 1806 Dr. John C. Warren became associated with his father, Dr. John Warren, in teaching anatomy and surgery, and succeeded to the full professorship on the death of the elder Warren in 1815.

John Collins Warren (1778–1856) was born in Boston, August 1st, 1778. He was the eldest son of Dr. John Warren, the founder of the Harvard Medical College. He graduated from Harvard College in 1797, and after a year's delay began the study of medicine. In 1799 he went to London and entered Guy's Hospital as a dresser

to Mr. William Cooper, senior surgeon to that hospital, who was soon after succeeded by Mr. Astley Cooper, his nephew. After a year's service he left London for Edinburgh, where he attended the lectures of Monroe and John and Charles Bell, and received his medical degree. He visited Paris, where he remained one year attending lectures, and then returned home in 1802. He was the founder of the Massachusetts General Hospital and one of its surgeons until his death. In 1806 he was associated with his father in the chair of anatomy and surgery, and on the death of the latter in 1815 he became full professor, and continued in that position until 1847, when a professorship of surgery was created, and he became the emeritus professor. He died May 4th, 1856.

Fig. 8. — John C. Warren (1778–1856).

He was educated in the traditions of the Edinburgh school and was influenced in his teaching and practice by the precedents of British surgery.

The successor of Warren in the Harvard school, in 1847, was Dr. George Hayward (1791–1863). He was a native of Boston, a graduate of Harvard College in 1809, but he graduated in medicine from the University of Pennsylvania in 1812. As a teacher, it is stated, "thoroughly versed in the principles and theory of surgery, he was a remarkably practical and popular teacher in the professor's chair and at hospital clinics." He died in 1863.

In 1849 Dr. Henry J. Bigelow succeeded Hayward as professor of surgery in the Harvard school, and continued in that position until 1882, a period of thirty-three years. Bigelow was qualified by birth, mental endowments, and preparatory training, not only to maintain the high standard of educational qualifications which the Harvard school required of its graduates, but to advance that standard so that it kept pace with the rapid development of the medical sciences during the period of his service. It is well stated of him that as "inventor and discoverer by nature, his constant aim was to enlarge the boundaries of his profession, and to this end his fertility in ideas and remarkable mechanical ingenuity came to his aid." He was pre-eminently a master of both the science and the art of surgery, and in his teaching he was able so to combine principles and practice that the student became proficient in both branches of the subject.

Fig. 9. — Henry Jacob Bigelow (1818–1890).

Henry Jacob Bigelow (1818–1890) was a native of Boston, and son of Dr. Jacob

Bigelow. He graduated at Harvard College in 1837, and received his medical degree from the same institution in 1841. In 1845 he began teaching surgery in the Tremont Street Medical School, and in 1849 he was appointed professor of surgery in the Harvard Medical College, a position which he held until 1886, a period of thirty-seven years. He was elected a member of the surgical staff of the Massachusetts General Hospital in 1846. He died in 1890.

This brings our narrative of the pioneer medical schools to the close of the *formative* period in the evolution of American surgery. They had laid broad and deep the foundations on which the character of its future practice was to be constructed. During the quarter of a century which has since elapsed these schools have been the master builders on those foundations, and to-day they are universally recognized as occupying a foremost position among the world's best institutions for medical instruction. Along the line of succession to the founders of surgical education in these first schools, we recognize the names of surgeons whose achievements in practice are the crowning glory of American surgery.

Not less illustrious in the annals of surgery are the names of many of the graduates who went forth from these schools, imbued with the highest ideals of professional character and animated by the adventurous spirit which pervaded all ranks of our young communities, to establish other schools at the centres of population. The promoters of the new colleges were more frequently enthusiastic young surgeons, whose ambition found its most natural expression in teaching others the science and art of their profession. We have not space to follow the development of these schools, nor would it be instructive to dwell upon their individual peculiarities further than is necessary to illustrate the American independence of precedents and the resourcefulness of their promoters, as seen in the organization of several of the earlier schools by graduates of the pioneer colleges.

In the year 1798 the Medical Department of Dartmouth College, at Hanover, N. H., was established, at the suggestion of Dr. Nathan Smith, a graduate of Harvard Medical College. The most interesting feature in the organization of this school was the composition of the faculty, which consisted of a single person, viz., its promoter, Dr. Nathan Smith. For twelve years he gave courses of lectures on all of the different branches of medicine then taught, except two courses in the department of chemistry.

Nathan Smith (1762–1829) was born at Rehoboth, Mass., September 30th, 1762. His education was obtained at the public school. At the age of twenty-four he witnessed a surgical operation, which so impressed him that he determined to study medicine, and accordingly applied to the surgeon to be admitted to his office as a student. He was directed to prepare himself for admission to Harvard College before commencing the study of medicine, which he promptly did, and was then allowed to enter and register as a student. After three years of study he located in practice at Cornish, Vt. Soon after, he attended the lectures on medicine and collateral sciences at Harvard College, from which he received the degree of Bachelor of Medicine in 1790. He returned to his practice, which he pursued with marked

success for five or six years. During this time he became much impressed with the low grade of educational qualifications of the practitioners with whom he was brought in contact. On this account his biographer states that "he was led to project a medical institution in connection with Dartmouth College, in order to rear up for the widespread regions of the interior of New England a race of better educated, more enlightened, and more skilful physicians and surgeons." His plans being approved by the president of the college, Dr. Smith sought to better qualify himself for the new duties by attending the schools of London and Edinburgh. He returned in September, 1797, and early in the year 1798 began a course of lectures which embraced the entire circle of the medical sciences, as then understood, and which he repeated for twelve successive years.

FIG. 10.—Nathan Smith (1762–1829).

In 1813 he was invited to the chair of "physic and surgery" in the recently established Medical Department of Yale College, which he accepted. He subsequently gave one course in Dartmouth College, one in Vermont University, and five in the Medical Institution of Bowdoin College at Brunswick, Maine. He died January 26th, 1829, in the sixty-seventh year of his age.

In estimating the character of Dr. Nathan Smith as a surgeon and teacher of surgery, we have the judgment of his colleague in the Yale Medical College, Prof. Jonathan Knight. He says: "For the duties of a practical surgeon, Dr. Smith was eminently qualified. . . . To these he brought a mind enterprising, but not rash; anxious, yet calm, in deliberation; bold, yet cautious in operation. . . . There was no formidable array of instruments, no ostentatious preparation; . . . all useless parade was avoided. . . . His whole mind was bent upon its performance."

"As an instructor," says Professor Knight, "the reputation of Dr. Smith was high, from the time he began the business of instruction. . . . That for many years he gave instruction upon all the branches of medical and surgical science, that this instruction was to classes of intelligent young men, and that many who were thus instructed have become eminent in their profession, prove not only versatility of talent, but variety and extent of information, with a happy method of communicating it. . . . He sought no aid from an artificial style, but merely poured forth, in the plain language of conversation, the treasures of his wisdom and experience. . . . His object was to instill into the minds of his pupils the leading principles of their profession, not entering fully into the details of the practice, but leaving it for them to apply these principles to individual cases as they should present themselves. These principles he would illustrate by appropriate cases furnished by a long course of practice, related

always in an impressive, and often in a playful manner, so as at once to gain the attention and impress the truth illustrated upon the mind. . . . He endeavored to inspire them, both by precept and example, with a love of their profession, with activity in the practice of it, and a zeal for its best interests."

Of the influence of Dr. Smith upon the profession of New England, Professor Knight remarks: "His influence over medical literature was equally extensive. This influence was exerted, through his large acquaintance among medical men, by his advice and example, as well as more directly through the medium of the various medical schools which were favored with his instructions. By means of his influence thus exerted, he effected, over a large extent of country, a great and salutary change in the medical profession. The assertion that he has done more for the improvement of physic and surgery in New England than any other man will by no one be deemed invidious."

From Dr. Nathan Smith's school at Dartmouth many students graduated who became reputable surgeons, and one, Dr. Reuben D. Mussey, achieved a national reputation, both as a surgeon and as an educator. Like that of his master, Mussey's early life was a continuous struggle to obtain an education and prepare for his future work. He was evidently adapted for pioneer duties, as he early undertook experimental research. While a student, he controverted Dr. Rush's theory of the non-absorbent power of the skin by a series of carefully conducted experiments. His thesis on this subject attracted much attention. His controversy with Sir Astley Cooper, in which he maintained that a fracture of the neck of the thigh bone within the capsule could unite by bone, illustrated Mussey's careful study and observation, and the tenacity with which he held his opinions. As a teacher his work was in the West, and chiefly in Cincinnati. He was the founder of the Miami Medical College.

Fig. 11.—Reuben D. Mussey (1780–1866).

Reuben Dimond Mussey (1780–1866) was a native of Pelham, N. H. His father was a physician, but the son had to earn the means to enable him to obtain an education. He graduated from Dartmouth College, in 1803, and became a pupil of Dr. Nathan Smith, and graduated in 1806. After three years of practice he attended the lectures in the University of Pennsylvania, from which he graduated in 1809. He located in Salem, Mass., and soon became prominent as a successful surgeon. He began to give lectures on different medical subjects, and in 1822 he was appointed professor of anatomy and surgery in the Dartmouth Medical School. In 1833–35 he lectured on anatomy and surgery in the Bowdoin Medical College and in 1836–37 he gave the course on surgery in the medical college at Fairfield, N. Y. In 1837 he was invited to professorships in New York City, Nashville, Tenn., and Cincinnati, Ohio.

He accepted the latter and became the professor of surgery in the Ohio Medical College, a position which he held fourteen years. He then organized the Miami Medical College, and remained connected with it until he retired from active duties, in 1858, and located in Boston. He died in 1866.

Gross speaks very disparagingly and even contemptuously of Mussey's personality and his power as a teacher. He says: "Mussey was of low stature, of an attenuated form, with high cheek bones, a prominent chin, a small gray eye, and ungraceful gait." As a lecturer he "was dull in the extreme. He was not only slow in his delivery, but deficient in his animation and in grace of manner. His words came forth tardily, as if he were in doubt as to their precise import or as to the construction that might be put upon them by his hearers." His lectures were not "learned, profound, or discursive, . . . for Mussey was not a man of reading." He adds: "If we may be able to credit those who professed to be able to judge of them and who had listened to other teachers on similar topics, his lectures must have been instructive." Probably Gross places the true estimate on Mussey's teaching in his conclusion: "His lectures owed their chief value to their practical adaptation to the daily and hourly wants of the practitioner."

The abilities of Dr. Nathan Smith as an educator and his activity in establishing new medical schools were transmitted to his son, Dr. Nathan R. Smith. For more than half a century as a teacher in many medical schools, and as an author and inventor, the latter exerted a marked influence upon the progress of scientific surgery. He was an attractive lecturer, a skilful operator, and an ingenious inventor. One of his most useful publications is his "Memoirs," in which he reproduced the substance of his father's teaching. His most important invention was an anterior splint for fractures of the thigh.

Fig. 12.—Nathan Ryno Smith (1797–1877).

Nathan Ryno Smith (1797–1877) was a native of New Hampshire. He graduated from Yale College in 1817, studied medicine under his father's direction, and received his degree from the Jefferson Medical College, Philadelphia, in 1820. He located in Burlington, Vt., and in 1825 founded the Medical Department of the University of Vermont, and was appointed its first professor of anatomy and surgery. In 1826 he was called to the chair of anatomy in the Jefferson Medical College, Philadelphia. In 1827 he became professor of surgery in the University of Maryland, Baltimore, and in 1828 accepted the chair of medicine in the Transylvania University, Lexington, Ky. After twelve years' service he returned to Baltimore as professor of surgery in the University of Maryland. He retained this position until 1870, when he resigned and became professor of clinical surgery. He died in 1877.

In 1817 the Medical Department of Transylvania University was established at Lexington, Ky., and the first opportunities were offered to obtain a medical education in the Southwest. The founder of this school was Dr. Benjamin W. Dudley, one of the most eminent surgeons of that period. There was no medical college west of the Alleghanies, and the need of facilities for medical instruction of a rapidly increasing profession in the great Southwest was very pronounced. Dr. Yandell, the historian of "Pioneer Surgery in Kentucky," remarks as follows: "The history of the Medical Department of Transylvania University . . . would practically cover Dr. Dudley's career, and would form a most interesting chapter on the development of medical teaching in the Southwest. . . . Dr. Dudley created the medical department of the institution and directed its policy."

The testimonies in favor of Dudley's qualifications as a teacher of surgery are numerous. One of his colleagues thus speaks of him as a professor: "He was magisterial, oracular, conveying the idea always that the mind of the speaker was troubled with no doubt. He was always, in the presence of his students, not the model teacher only, but the dignified, urbane gentleman; conciliating regard by his gentleness, but repelling any approach to familiarity, and never, for the sake of raising a laugh or eliciting a little momentary applause, descending to coarseness of expression or thought. So that to his pupils he was always and everywhere great. As an operator they thought he had distanced competition. As a teacher they thought he gave them not what was in the books, but what the writers of the books had never understood. They were persuaded that there was much they must learn from his lips or learn not at all."

Benjamin Winslow Dudley (1785–1870) was born in Virginia, April 25th, 1785. When one year of age his father removed to Lexington, Ky., where he was reared. His opportunities for obtaining an education were very meagre. He studied medicine in the office of a physician and received his degree from the University of Pennsylvania two weeks before he was twenty-one, in 1806. Ambitious of success as a surgeon, he determined, after two or three years of practice, to visit the hospitals abroad. Not having sufficient means at his command, he purchased a flat-boat, loaded it with produce, and took it to New Orleans, where he exchanged it for flour. This cargo he took to Europe and sold at a considerable advance, and with the proceeds prosecuted his surgical studies for a period of four years in the hospitals of Paris and London. He returned to Lexington in 1814, and rapidly acquired reputation as a surgeon. In 1817, largely through his influence, the Medical Department of Transylvania University was established, and Dr. Dudley was appointed the professor of anatomy and surgery. He died in 1870.

Fig. 13.—Benjamin Winslow Dudley (1785–1870).

In 1826, Jefferson Medical College, of Philadelphia, was founded by Dr. George McClellan, a graduate of the Medical Department of the University of Pennsylvania. McClellan was a type of the aspiring and aggressive young surgeons of that early period. He had been a pupil of Dorsey, the assistant of Physick, a brilliant lecturer and accredited author. Soon after his graduation McClellan began teaching anatomy and surgery, and his vivacity of manner and fluency of speech attracted large classes. It is stated that as a public teacher his style was purely extemporaneous; he became so absorbed with his subject as to be unconscious of those around him. His lectures achieved a popularity and produced an effect seldom equalled. As a practical surgeon he took rank with the most successful practitioners of that day. The school which he founded has been one of the largest contributors, in its graduates, to the ranks of eminent practical surgeons and teachers.

Fig. 14.—George McClellan (1796–1847).

George McClellan (1796–1847) was born at Woodstock, Conn., on the 23d of December, 1796. He was of Scotch descent. He prepared for college at an academy in his native town, and entered the sophomore class of Yale College at the age of sixteen. On his graduation in 1815 he began the study of medicine in the office of Dr. Thomas Hubbard, of Pomfret, afterward professor of surgery in the Medical School at New Haven. In 1817 he attended lectures in the Medical Department of the University of Pennsylvania, and entered the office of Dr. John Syng Dorsey, then professor of materia medica and anatomy in the university. In 1818 he became a member of the resident staff of the Philadelphia Almshouse. He located in Philadelphia and began to give private lectures on anatomy. In 1825, in co-operation with friends, he obtained a charter for the Jefferson Medical College, in which he occupied the professorship of surgery until the year 1838, when the professorships were all vacated by the trustees and a new organization formed, from which Dr. McClellan was excluded. He applied at once to the legislature for a charter of another college, which was granted, and "The Medical Department of Pennsylvania College," at Gettysburg, was established. The lectures were commenced at Philadelphia in November, 1839. At the close of the fourth annual course of lectures the faculty resigned, owing to pecuniary complications, and Dr. McClellan retired to private practice. He died suddenly, of perforation of the colon, on the 8th day of May, 1847.

One of the most illustrious surgeons and educators of our period was a private pupil of McClellan, a graduate of the Jefferson Medical College, and his successor to the chair of surgery—Dr. Samuel D. Gross. It is impossible to estimate the vast influence of Gross upon the character of surgical practice during his long career of over half a century, and in the threefold capacity of an original investi-

gator, a popular teacher in many schools, and an accepted authority on general surgery.

Samuel David Gross (1805–84) was born near Easton, Pa., July 8th, 1805. He received a classical education and for two years was a pupil of Dr. George McClellan, graduating in medicine from Jefferson Medical College in 1828. He located in Philadelphia, and in 1830 published a work on "Diseases and Injuries of the Bones and Joints." In 1833 he became demonstrator of anatomy in the Medical College of Ohio, and in 1835 he was appointed professor of pathological anatomy in the Medical Department of the Cincinnati College. His lectures were the first delivered in this country on that subject, and resulted in the preparation of a work on the "Elements of Pathological Anatomy," the first work of the kind in the English language. In 1839 he was appointed professor of surgery in the University of Louisville, Ky., and in 1850 he was appointed professor of surgery in the Medical Department of the University of the City of New York. He gave but a single course of lectures in New York, and returned to his former position in the Louisville school. In 1865 he was appointed professor of surgery in the Jefferson Medical College, Philadelphia, from which he retired in 1882. He died in 1884.

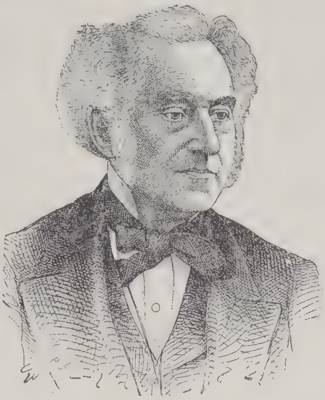
Fig. 15.—Samuel David Gross (1805–1884).

Dr. Isaac M. Hays has given the following graphic but truthful description of Gross as a man and as a teacher: "Dr. Gross's magnetic form and dignified presence, his broad brow and intelligent eye, his deep, mellow voice, and benignant smile, his genial manner and cordial greeting remain indelibly impressed upon the memory of all who knew him. He was a man of deep mind and broad views, and he was a model of industry and untiring zeal. . . . His style was vigorous and pure. It is safe to say that no previous medical teacher or author on this continent exercised such a widespread and commanding influence. . . . His writings have been the most learned and voluminous and his classes among the largest that have ever been collected in this country."


Fig. 16.— Daniel Brainard (1812–1866).

In 1837, Rush Medical College, of Chicago, was founded by Dr. Daniel Brainard. This was the pioneer medical school in the Northwest, and has always maintained a high grade of surgical instruction.

Daniel Brainard (1812–66) was a native of Whitesborough, N. Y. He was educated at the public schools and studied medicine in the office of the village physician. He attended two courses of lectures at the Medical

College at Fairfield, N. Y., but graduated from the Jefferson Medical College, Philadelphia, in 1834. He began to give lectures on anatomy and surgery in the Oneida Institute, and in 1836 removed to Chicago. He went abroad in 1839 and returned in 1841, when he was appointed professor of anatomy in the University of St. Louis, Mo. He took an active part in organizing the Rush Medical College, which was chartered in 1837, and opened in 1843, Brainard occupying the chair of surgery. He died in 1866.

Brainard began teaching at an early period of his professional career, and thus qualified himself to be an educator. He followed the trend of enterprising young men of that day, and sought fame and fortune in the far West, locating in Chicago. In this growing city and future metropolis Brainard found an ample field for the exercise of his talents as a surgeon and, finally, as an educator. He became eminent as a practical surgeon, and in the establishment of Rush Medical College he found his opportunity as a teacher. In the latter capacity we have the testimony of his biographer that: "As a teacher he stood without a rival."

The pioneer teacher of surgery in the extreme South was Dr. Warren Stone (1808–72), of New Orleans. He was a native of Vermont and studied medicine under Dr. Amos Twitchell, one of the most famous surgeons of that day in this country. He took his medical degree at Philadelphia in 1825, and located in New Orleans. He was connected with the Medical Department of the University of Louisiana from its organization in 1834. During the first and second sessions he discharged the duties of demonstrator, and was appointed surgeon to the Charity Hospital. In 1836 he was appointed lecturer on anatomy, and in 1837 professor of that branch, at the same time giving the lectures on surgery. In 1839 anatomy was separated from surgery, and he assumed the full duties of the chair of surgery. For upward of a third of a century Stone taught large classes of students and exercised a great influence upon the practice of surgery. Gross attributes his success to his large heart and the native powers of his mind, strong and well poised.

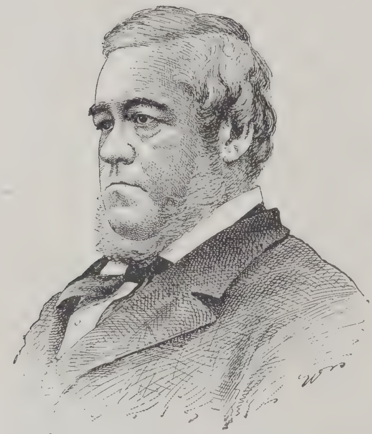

Fig. 17.—Warren Stone (1807–1878).

One of the most conspicuous surgeons of the South, who at an early period took an active part as a teacher in the newly organized medical schools, was Dr. Paul F. Eve. He was a native of Georgia, a graduate of the University Medical College, Philadelphia, and later an attendant upon the lectures and practice of the leading surgeons of London and Paris. He began teaching in 1832, in a small college in Georgia, and from that time until his

death he was engaged in giving courses of lectures on surgery in a large number of colleges. On his final settlement in Nashville, Tenn., he became eminent as a practical surgeon. As an instructor he was popular and had flattering offers of professorships of surgery in the older colleges.

Paul Fitzsimmons Eve (1806–78) was a native of Augusta, Ga., born in 1806. He graduated at Franklin College, Athens, Ga., in 1826, and received his medical degree from the University of Pennsylvania in 1828. After an absence in Europe of three years, he returned and was appointed professor of surgery in the Medical College of Georgia, 1832, recently organized. In 1850 he was appointed to the chair of surgery in the University of Louisville, as successor to Gross, but retained the position only one session, when he accepted the professorship of surgery in the University of Nashville. In 1868 he was called to the chair of surgery in the Missouri Medical College, St. Louis, but after two courses of lectures he returned to Nashville and became professor of clinical and operative surgery in the Nashville Medical College, then being organized. He had invitations to accept professorships in colleges in New York, Philadelphia, New Orleans, and Memphis, but he declined them and remained in Nashville until his death in 1878.

Fig. 18.—Paul Fitzimmons Eve (1806–1878).

The founder of the St. Louis Medical College, Missouri, was Dr. Charles A. Pope, one of the most prominent surgeons of the West at that period.

The establishment of a medical school on the Pacific Coast was effected by Dr. Cooper, a surgeon having a wide reputation for his skill as an operator and his enterprising spirit.

We cannot farther trace the progress of the schools which were to educate the future surgeons of this country in the principles of scientific surgery and to illustrate by precept and practice its art, but must notice other educational forces which have more or less effectively impressed a national stamp upon the American practice of surgery. First, and most important, is the development of clinical instruction as a necessary qualification of the surgical student on his graduation.

The value of *clinical instruction* was recognized and efforts were made to supply it by the medical officers of the Pennsylvania Hospital, as early as 1765. Clinical lectures were subsequently given in the almshouses of Philadelphia and New York, and later in the New York Hospital and the Massachusetts General Hospital. There arose, however, two serious difficulties that obstructed the progress of this most important improvement in medical education. In the first place, there was early developed an intense prejudice on the part of lay managers of hospitals against the exposure of patients to the observation of medical

students and to the public discussion of their ailments. It was believed that the inmates would regard such exposure as an outrage upon common decency and universally rebel against the practice. Happily, experience proved, not only that patients did not resent such treatment, but that they were always gratified with being selected as the subjects of special attention and study, while those who were passed by complained of neglect. Again, the fact developed that those hospitals were most efficiently and carefully supervised in the medical and surgical service where the visiting staffs gave clinical instruction. The result of these experiences has been, not only the removal of all prejudices against clinical teaching in hospitals, on the part of the public, but a disposition of managers to encourage the medical schools to use the service for the purpose of teaching. In the second place, there was a class of teachers who were opposed to hospital attendance by medical students until they had regularly graduated from the schools. It was alleged that undergraduates could not be benefited by attending lectures on subjects which they could not by any possibility understand, and about which they were liable to obtain false views that would prove very detrimental in practice. Experience, however, established the fact that the most thoroughly qualified graduate in both technical and practical knowledge was the student who had received clinical instruction from the outset of his course of study.

Prior to 1861 clinical instruction was, however, little more than an interesting incident in the life of the medical student. He visited the hospitals to witness an advertised operation in the interval of lectures, rather for the relaxation and excitement which the occasion afforded than for any positive knowledge he expected to acquire. But a most important change in medical education was impending. Clinical teaching was to become an essential part of the system of instruction, and attendance upon its lectures was no longer to be a pastime, but a compulsory duty with every aspirant for graduation.

This advance in medical education was largely due to Dr. James R. Wood, of New York. On the reorganization of Bellevue Hospital, New York, the visiting staff of physicians and surgeons, under his guidance and direction, began to give systematic courses of clinical instruction to the medical students of the several colleges. The staff included young men of marked ability, ambitious of success as teachers, and animated with that genuine enthusiasm which stimulates students to high endeavor. Dr. James R. Wood, Dr. William H. Van Buren, Dr. John T. Metcalfe, Dr. John O. Stone, Dr. Benjamin W. Macready, Dr. Lewis A. Sayre, Dr. George T. Elliott were lecturers who always attracted large classes. So popular had these lectures become during the years 1855–60, that it was determined to organize a chartered medical college, in which clinical instruction should form part of the prescribed course of study. Bellevue Hospital Medical College began its career in 1861, with the avowed purpose of combining didactic and clinical instruction. The popularity of the clinical teaching in the hospital gave the new college immediate success.

James Rushmore Wood (1813–82) was born in the city of New York, on the 14th day of September, 1813. He had but limited opportunities for education at the Friends' Seminary, in New York City. He began the study of medicine in 1829 with Dr. David L. Rogers, and attended his first course of lectures at the College of Physicians and Surgeons, in 1831. He graduated in 1834 from the Medical College at Castleton, Vt., and was soon after appointed demonstrator of anatomy in that College. He located in New York and rapidly advanced to an influential position in the practice of surgery. He became connected with the management of Bellevue Hospital, then an almshouse, and in 1847 he effected a complete change in the organization of that institution, by converting it from an almshouse, under a resident physician, into a hospital, with its visiting and resident staffs of physicians and surgeons, and under the direction of a medical board. He began a systematic course of clinical instruction, which drew large numbers of students to its wards and led to the creation of the Bellevue Hospital Medical College in 1861. Wood was appointed professor of operative surgery and surgical pathology in the new school, a position which he held upward of fifteen years, and on retiring was appointed emeritus professor. He died in 1882.

Fig. 19.—James R. Wood (1813–1882).

Dennis, the biographer of Wood, states that he "was foremost in the view that medicine is a science pre-eminently of demonstration as well as of observation, and it was the union of clinical and didactic teaching that in his opinion best attained the object of medical education."

Hospital instruction in the practice of surgery has become increasingly important in these later years, when anæsthesia and antisepsis have given to the technique of operations a scientific precision, even to the minutest details of the preparation of the patient, the operator, his assistants, the room, the appliances, the administration of the anæsthetic, the immediate aid of the surgeon, and, finally, the preparation and application of the permanent dressings. The high-grade technical operator regards each of these innumerable details as vitally essential to the success of the operation. But it is impossible to gain the requisite expertness in the manipulation of these complex details except under the conditions which are enforced in a modern hospital. The hospitals of this country, therefore, now so numerous and well equipped, have become essential factors in the education of surgeons for practical duties. Hundreds of surgeons graduate annually from our hospitals, fully qualified by education and practice to undertake the most responsible duties of their profession.

But the modern hospital not only serves as a school for perfecting the young surgeon during the period of his education in the manual or art of surgery, but it supplies conditions nowhere else obtainable, which enable the surgeon to apply

those arts in practice with almost absolute success. In other words, the modern well-equipped hospital is essential to the highest degree of success in the practice of surgery, whatever may be the skill and experience of the individual surgeon. Within the walls of thousands of hospitals in this country are found ready and awaiting his order every condition, thing, or circumstance which the surgeon can possibly in any emergency require for successful practice. And these hospitals are increasing at a rate that positively insures to every community the opportunity of having every variety of disease or injury, amenable to surgical treatment, immediately placed under conditions most favorable for recovery. Not only are public hospitals increasing in such vast numbers, but on every hand surgeons are establishing their own private hospitals, equipped with everything required for the most successful work. To these should be added the increasing number of large private corporate hospitals, where operations are daily performed by the score, with an accuracy in all the details of the procedure comparable to that performed by instruments of precision. Large numbers of these hospitals are devoted to special classes of diseases, as the eye and ear, the throat and nose, the genito-urinary apparatus, and in each will be found the highest grade of surgical practice. Some of these corporate hospitals are devoted to general surgical practice, where operations are performed on a vast scale and with marvellous success. Finally, there are corporate hospitals which combine not only all the specialties with general surgery, but they are supplied with laboratories for biological and pathological investigations, and appliances for every form of mechanical and instrumental treatment. These great institutions, which are rapidly increasing in various parts of the country, indicate that the time is approaching when the American practice of surgery, in all its details, will be established on strictly scientific principles.

"*The Training-school for Nurses*" in the modern hospital, inaugurated in 1872 in Bellevue Hospital, New York, is a factor in the successful practice of surgery, the value of which it is quite impossible to estimate. That these schools have revolutionized practice is the universal testimony of both physicians and surgeons. Only the operator himself can, from his individual experience, appreciate at its full value the assistance of the expert and reliable nurse, who prepares his patient, deftly meets every want and emergency during the operation, and during the critical hours or days or weeks of convalescence faithfully watches every symptom, rightly interprets its meaning, whether for good or for evil, and promptly and intelligently applies the prescribed remedial measure. So essential has the trained nurse become to success in the practice of surgery in this country that every hospital, however small, has its corps of nurses, and no surgeon will operate without their aid when it can be obtained.

It is often alleged that our system of medical education is very defective in the advantages of pathological research which are afforded by large *museums*.

The few morbid specimens which individual surgeons were able to save in their practice threw but little light on obscure questions of pathology, and it has been believed that only in the great museums of Europe can such studies be adequately pursued. Efforts to supply this want have been made by individual surgeons, notably by Dr. James R. Wood, of New York, and Dr. Thomas D. Mütter, of Philadelphia. But there is much truth in the conclusion of Hamilton, who consulted all of the great museums of the world, while preparing his work on "Fractures and Dislocations," to determine positively doubtful questions: "Nothing is more unreliable than the testimony furnished by cabinet specimens whose clinical history is wholly unknown, and in reference to which in many cases it is impossible to say whether their present condition was due to traumatism before or after death, or, indeed, whether it was not due to some long-preexisting pathological cause."

In place of the museum the colleges have now their well-equipped laboratories and their courses of instruction in the closely allied branches of a complete medical education, viz., biology and pathology. In these departments the student has immediate access to the healthy and morbid specimens, so freshly prepared as to be wellnigh living in their accuracy of illustration, with necessary demonstrations of all doubtful questions by the instructor. As a means of firmly implanting in the student's memory useful practical facts, the present method of teaching these subjects in the laboratory has many and important advantages over the mere study of museum specimens.

The *literature* of a profession is not only a safe guide to the estimation of the scientific spirit which inspires its practice, but it is an important educational force in developing the character of the coming generations of practitioners. In this view the literature of American surgery deserves proper estimation. Its development has necessarily kept pace, both in quantity and quality, with the progress of the schools in raising the standard of medical educational qualifications. From one book in a quarter of a century it has increased to a score of books in one year, and from one serial publication it has multiplied to fifty periodicals. It began, both in book and in serial form, during the last quarter of the eighteenth century, and was for a considerable period but little more than a transcript of British surgical literature. As such, however, it shows a wise and judicious discrimination on the part of authors to meet the wants of the practising surgeon. But, in time, books and even articles were published, which on account of their originality were epoch-making in their influence.

The author of the first surgical work was Dr. John Jones, of New York, and it was written to meet the emergency which confronted the medical profession at the opening of the war of the Revolution. Dr. Jones was well qualified for this task. He had been educated in the British and French schools, had practised surgery with great success in New York for a score of years, had given full

courses of lectures on surgery in the Medical Department of King's College for seven years. His special qualification for this task grew more directly out of his experience in the war on the Canadian frontier, between the English and French, in 1755, where he won distinction as a surgeon. The remarkable case of the French general, Baron de Dieskau, who was wounded and taken prisoner and placed in charge of Jones, illustrates his skill as a military surgeon. The general was wounded in the hip, in the thigh, in both knees, and through the pelvis, the latter wound involving the urinary bladder, so that urine escaped from the wound of entrance and of exit. Though the conditions under which the patient was treated were most unfavorable, he recovered so as to be able to return to Europe.

Dr. Jones's work was entitled "*Plain, Concise, Practical Remarks on the Treatment of Wounds and Fractures.*" It was printed at New York, in 1775, but in 1776 a second edition was issued at Philadelphia, to which was added the popular work of Van Swieten on "*The Diseases Incident to Armies and Gunshot Wounds.*" The work was what its title announced—simply *plain, concise,* and *practical remarks* on all that at that time was known of military surgery. The merits of the book lie in its adaptation to the wants of the surgeons of the Continental Army, few of whom had any useful surgical knowledge, theoretical or practical. The only works on surgery at the time were meagre treatises, and even these were accessible to but few. The appearance of Dr. Jones's work, in small manual form, at the very beginning of the war, was an achievement of national importance. Dr. David Ramsay, a contemporary medical historian, says this work "will long remain a monument both of professional skill and patriotism of its author."

Notwithstanding the progress of the schools and the great impulse that had been given to the study of scientific surgery by Hunter's teachings, especially among American students, nearly forty years elapsed after the issue of Dr. Jones's work before another native surgical treatise appeared. In 1813 Dr. John Syng Dorsey, of Philadelphia, published a systematic work, entitled "*Elements of Surgery,*" in two volumes, 8vo, which reached a second edition in 1818, and a third in 1823. It was a work of great merit for that period, as it faithfully illustrated the practice of British surgery, but, in addition, it gave publicity to Physick's surgical teachings, which might otherwise have been lost to surgeons. The value of this work was recognized by the Edinburgh school, which adopted it as a text-book.

In 1824 appeared "*The Institutes and Practice of Surgery,*" by Prof. William Gibson, professor of surgery in the Medical Department of the University of Pennsylvania, in two volumes. It was announced to be "outlines of a course of lectures," and "published at the request of students who want a text-book"; the "work must be considered as a mere *outline* of the lectures, to be filled up by numerous illustrations, chiefly models, morbid preparations, *magnified* draw-

ings, and *imitations* on the dead subject. The last two modes of instruction I consider peculiarly my own." That this work was well received we learn from the preface of the second edition, which appeared in 1827: "The praises which have been bestowed on the work by European and American critics, though far beyond, in many instances, any merit I should be entitled to claim," etc. In the preface to the third edition, which appeared in 1833, the author says: "This work has been pronounced by hypercritics a book on the practice of medicine." He adds: "A greater compliment could not have been paid to it, and yet it argues a very narrow view on the part of those who strive to affix limits to sciences which blend and often unite in every possible way." He defends this feature of his work on the ground that practitioners in this country must practise both medicine and surgery. That the work was well adapted to the wants of the profession is evidenced by the appearance of a fourth edition in 1835 and a fifth edition in 1838.

In 1859 appeared "*A System of Surgery*," by Prof. S. D. Gross, professor of surgery in Jefferson Medical College, in two large volumes. The author says: "The object of this work is to furnish a systematic and comprehensive treatise on the science and practice of surgery, considered in the broadest sense. . . . My aim has been to embrace the whole domain of surgery and to allot to every subject its legitimate claim to notice in the great family of external diseases and accidents." He continues: "It may safely be affirmed that there is no topic, properly appertaining to surgery, that will not be found to be discussed to a greater or less extent in these volumes." This system of surgery was a work of very unequal merit, owing to the treatment of such a wide range of subjects by a single author, but it became the text-book of the schools and retained that position through many editions.

Although the works of several British surgical authors appeared and were republished in this country during this period, the native works of Dorsey, Gibson, and Gross were generally accepted as text-books and guides to American practice. In addition to these works on general surgery, several treatises on special branches of practice were published and deserve notice. As the authors of these works were surgeons educated in the home schools, the text illustrates the stage of progress in the practice of these specialties to which they had attained.

In 1851 Gross published a work entitled "*A Practical Treatise on the Diseases and Injuries of the Urinary Bladder, the Prostate Gland, and the Urethra.*" This treatise has the merit of being the first complete work in the English language on these organs. At the time of its appearance several British surgeons had written monographs on these subjects, but no one had ventured to cover the whole field as did the American author. The only other accessible complete treatise of the kind was that of the French authority, Civiale. In his preface Gross states that his sole object "has been to furnish a monograph on the dis-

eases and injuries of the urinary organs that should be worthy of the favorable consideration of his professional brethren and of the present state of medical science in this country." This work greatly improved the treatment of genito-urinary diseases and laid the foundation of that specialty as it is now recognized in the schools.

In 1860 Dr. Frank H. Hamilton published his work, entitled, "*A Practical Treatise on Fractures and Dislocations.*" It was the first treatise on these subjects written in the English language and supplied a pressing want. Hamilton was amply qualified for the task which he undertook. He had been a teacher of surgery from his graduation, in several colleges, and had a large experience as an expert witness in suits against physicians, so frequent at that time. The subject of litigation was usually malpractice in the treatment of fractures. The great diversity of opinions among surgeons at these trials and the entire absence of any reliable authority was the incentive that prompted him to undertake the investigations which form the basis of this treatise. He was indefatigable in the pursuit of facts, and endeavored, by experiments and personal visits to pathological museums in this country and Europe, to verify every statement and judicially establish every opinion which he should record.

Frank Hastings Hamilton (1813–86) was born at Wilmington, Vt., September 10th, 1813. He received his classical education at Union College, Schenectady, N. Y., and graduated in medicine from the Medical Department of the University of Pennsylvania in 1833. He located at Auburn, N. Y., but removed to Rochester, and in 1848 to Buffalo, and in 1862 to New York. He gave courses of lectures on surgery at the Pittsfield Medical School, Mass., and at the Geneva Medical College, N. Y. On the establishment of the Medical Department of the University of Buffalo he was appointed professor of surgery, a position which he held until his removal to New York. He gave courses of lectures in the Long Island Medical College, and was appointed professor of the specialty, "Fractures and Dislocations," in the Bellevue Medical College, New York, in 1862. Soon after, he enlisted as brigade surgeon and served in the Army of the Potomac, acting as medical inspector in 1863, but ill health compelled his resignation. In 1875 he resigned his professorship in the Bellevue Medical College. He died in New York, August 11th, 1886.

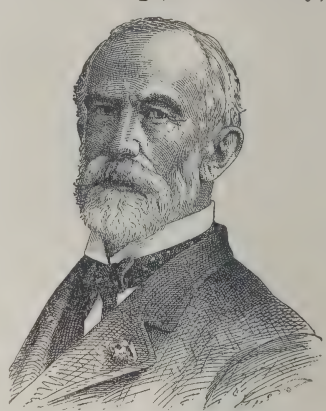

Fig. 20.—Frank Hastings Hamilton (1813–1886).

In the preface to the seventh edition of the treatise, published in 1884, the author reveals in apologetic terms the conscientious and judicial spirit in which the work is written: "From the beginning of his studies the author has found one of his most difficult labors in attempting to eliminate from the branch of science which he has undertaken to teach the numerous 'false facts' or un-

reliable statements derived from these several sources, and this must be accepted as his apology for his repeated expressions of scepticism in reference to testimony, some of which has been accepted, as is believed without sufficient examination, by writers whose opinions might be regarded as of more value than his own."

Its thoroughly scientific character, its accurate historical review, its large range of well-digested facts, its careful analysis of current theories and opinions, and its pure English style placed it at once among the classics of surgical literature. Although out of print, it still maintains its position among surgeons as the most reliable authority in the English language on fractures and dislocations.

Hamilton's work led to the establishment of a "chair of fractures and dislocations" in the Bellevue Hospital Medical College.

In 1861 Dr. Freeman J. Bumstead, of New York, published a work, entitled, "*Pathology and Treatment of Venereal Diseases.*" The object of the author was to furnish the student a full and comprehensive treatise on the venereal diseases, and the practitioner a plain, practical guide in their treatment. The work of Bumstead was received with great favor, and created so much interest in venereal diseases that the medical schools began to introduce courses of instruction on this subject as a specialty. Several editions of this work appeared, and during the lifetime of the author it maintained its position as the most complete and reliable work on venereal diseases in the English language.

During this period large numbers of monographs appeared on surgical subjects, some of which were of a high order of merit and greatly improved methods of practice. Several of these publications will be noticed in other sections of this paper.

In his historical sketch of the medical literature and institutions of this country, Dr. John S. Billings, an eminent authority, remarks: "Since the year 1800 medical journalism has become the principal means of recording and communicating the observations and ideas of those engaged in the practice of medicine, and has exercised a strong influence for the advancement of medical science and education." That the medical profession of this country has improved this method of advancement, is shown by his summary of medical journals published down to 1876, the number being one hundred and ninety-five, including reprints of foreign journals, making in all sixteen hundred and thirty-seven volumes. It is the universal testimony of surgeons that they have derived more benefit in the details of practice from the current information furnished by medical journals than from text-books of surgery.

The growth of medical libraries in this country is another striking feature of the evolutionary process by which the practice of surgery has advanced to a more and more nearly perfect state of development. In these latter days there is a flood-tide of surgical publications. The pioneer surgeons complained that they had no books to consult and had to rely on their own unaided judgment in the emergencies of practice; but modern surgeons have such a surplus that

they are compelled to adopt the co-operative plan, so popular in business circles, of forming libraries capable of accumulating all of the current surgical literature, for the use and common good of surgeons of every grade. These libraries are becoming more and more the great centres of education of the entire profession, and their influence in elevating the grade of practice becomes daily more and more evident. There are now 164 medical libraries in the United States, containing a total of 912,330 volumes.

With this review of the development of the educational qualifications of American surgeons, we are prepared to estimate the value of that education, as illustrated in the performance of the practical duties of their profession. We can select only the more important questions in the practice of British surgery at the beginning of our period, and consider their treatment by American surgeons. This examination will bring prominently into view the special characteristics of the American practice of surgery.

Mr. Erichssen, in his "Impressions of American Surgery," already referred to, remarks: "The bent of the mind of the American surgeon is, like ours, practical rather than scientific." There is ample proof that the achievements of American surgeons are to be found in the field of practice rather than in the laboratory. This fact is not due to a lack of interest on the part of American surgeons in the truths of science nor to failure to appreciate their value when applied to practice, but rather to the social conditions under which the surgeon begins his career. The community in which he locates is young, compared with those of the old world, and professional and business success is popularly estimated by those activities which have the greatest publicity. Whatever may be the qualifications of a graduate of one of our medical schools for a successful career as a scientist, if he has had the training of a practical surgeon in a modern hospital, he will almost invariably be so fascinated by the glamour of operations as to subordinate science to practice.

The American practice of surgery has always been characterized by self-reliance and resourcefulness. This has been due in part to the more limited means and agencies at the command of the practitioner in this country, and in part to the adventurous spirit which has always inspired every department of American activity. Thousands of surgeons have been compelled to practise their profession far removed from access either to the aid or the advice of a competent surgeon or to necessary instruments and remedial agents. Many new and difficult operations have been performed under these conditions and with a remarkable degree of success. It is true that operations under such circumstances are liable to have no scientific value, unless they incidentally suggest or reveal important facts hitherto unknown; but they do demonstrate, as no other method can, the boldness and daring of the operator and his mental equilibrium and resources in emergencies. A review of the practice of surgery in the early periods

of our national history proves that these attributes have always been dominant features of the qualifications of American surgeons.

As we have already stated, the practice of surgery in this country illustrated the progress and growth of British surgery transplanted to a virgin soil. Educated in all the traditions of the foreign schools, but unhampered by them in his practice and usually left to his own resources, the pioneer American surgeon was compelled to resort to new and untried, and even unheard-of methods, to meet emergencies. On this account the practice of surgery in this country has necessarily been characterized by a freedom from arbitrary and often impracticable rules, which have a controlling force with surgeons of the older countries. It has been very frequenty asserted that this freedom from technical rules in the practice of surgery is liable to result in dangerous adventures on the part of the surgeon, quite incompatible with judicious conservatism. But such has not been our experience; on the contrary, this very freedom has developed such an overpowering sense of personal responsibility that the surgeon has proceeded with a degree of caution the equivalent of true conservatism.

The evolution of the practice of American surgery necessarily kept pace with the progress of the medical schools in developing the educational qualifications of the future surgeons of the country. Prior to the year 1800, the three pioneer medical colleges had graduated too few students to have exerted any marked influence upon the profession at large, especially in regard to the practice of surgery. But during the first quarter of the nineteenth century the number of medical colleges rapidly increased and the grade of teaching greatly improved. The result appears in the increased activity of surgeons in performing formidable operations and the independence which characterized their departure from rules established by the foreign schools. It is during this period that we begin to trace the line of cleavage between American and European surgery, and from this time we more and more frequently meet the word "American" in surgical literature, in connection with new inventions and methods of operation. It was, therefore, during the early years of the nineteenth century that the evolution of what may properly be termed "the American practice of surgery" began to appear, and it is from that period we shall begin to trace its development and illustrate its distinctive features.

The treatment of aneurism was a subject of absorbing interest to British surgeons at the close of the eighteenth century. Hunter had perfected Anel's method of ligating the artery on the proximal side of the tumor, and had established the following principles: 1. The ligature should be applied at a sufficient distance from the tumor to insure a healthy condition of the artery. 2. The artery should not be disturbed more than is necessary to secure the passage of the ligature. 3. One ligature is sufficient. 4. The wound should be healed by first intention as far as possible.

Hunter's operation was performed with indifferent success by British surgeons, according to Home, owing to modifications which they made of the procedure of the original operator. The surgeons of the continent ignored this method of treating aneurism, chiefly because it had a British origin. But there was present at Hunter's first operation a young American surgeon from the city of New York, who thoroughly comprehended the opinions of the operator, and appreciated at its full value the immense importance of the operation. Dr. Wright Post was a pupil of a member of the staff of St. George's Hospital at the date of Hunter's first operation, which was performed in that hospital in December, 1785. Post returned to New York in 1786, and soon took a high rank as a teacher of anatomy and surgery. The treatment of aneurism by the new operation was evidently the theme of some of his lectures, for Mott, his most eminent student, says Post expressed the opinion that not only one carotid artery might be ligated for aneurism safely, but that both might be interrupted by ligature on the same person without harm, long before Astley Cooper operated on that artery.

Post's first operation was the ligation of the femoral artery for aneurism, in 1796. The patient had a femoral aneurism caused by a wound of the artery fifteen years previously. The precise location of the aneurism is not given, nor the point at which the ligature was applied. The patient recovered in the usual time and the tumor gradually diminished until it was reduced to a size not exceeding one inch in diameter. An interesting feature of the case was a continuance of the pulsation of the tumor, which Post attributed to the increased size of the anastomosing vessels due to the long continuance of the aneurism. The limb became as useful as it was before the accident.

This was the first operation for the cure of aneurism on the Hunterian principle in this country, and the beginning of the operator's career as the practical exponent of the Hunterian method of treating aneurism.

The first operation of ligating the common carotid for aneurism in this country was performed by Post, January 9th, 1813. The tumor was situated below the angle of the jaw on the right side, and measured six inches in length, four inches in breadth, and two inches in height. Two ligatures were applied and the artery was divided between them. The case did well and was discharged at the end of four months. The patient returned in two months, the tumor being large and fluctuating. It soon after opened and there was a hemorrhage of thirty ounces. It opened in a second place and discharged pus and blood; severe hemorrhage occurred several times, and once the patient was thought to have lost two quarts. Extensive suppuration occurred at the site of the aneurism, but the patient finally recovered.

The peculiarities of this operation were: (1) The passage of two ligatures around the artery, about three-quarters of an inch apart; (2) the passage of the ligature through the artery to prevent its slipping from the end of the cut ar-

tery, as recommended by Dionis and Cline; and (3) the division of the artery between the ligatures. The danger of hemorrhage from the slipping of the ligature from the cut end of an artery was at that time regarded as very great, and to prevent it the needle was placed on the ligature after it was tied, and the thread was passed through the artery close to the ligature and tied with the knot already made.

One year later, January 4th, 1814, Post applied a ligature to the external iliac for inguinal aneurism. It was the second operation on the artery in this country, Dorsey, of Philadelphia, having operated in 1811 successfully. The important feature in Post's case was the necessity of opening the peritoneal cavity to reach the artery, and the recovery of the patient. It is stated in the report that the strength and thickness of the peritoneum were considerably greater than natural, and its adhesion to the ligament so firm that the separation, which is ordinarily so easily effected, was found in this case altogether impracticable. To arrive at the artery, therefore, under these circumstances, it was necessary to cut through the peritoneum, and thus "expose the patient to the additional hazard of inflammation of this membrane, to which it is generally supposed to be very liable when an opening is made into the common cavity of the abdomen." But to accomplish this object there was no alternative, nor did Post hesitate in proceeding with the operation in this manner.

On the 28th of November, 1816, Post again ligated the common carotid artery for a pulsating tumor of the neck. The patient recovered from the operation, but died two years later, and the autopsy disclosed a tumor with no indications of a previous aneurism. It was to this case that Mott often alluded in his lectures, illustrating the difficulties of correctly diagnosing an aneurism from an abscess or solid tumor overlying an artery. At the consultation Post diagnosed the tumor as an aneurism, Stevens as an abscess, and Mott as a solid tumor; Stevens suggested to Post the propriety of exploring it by puncture, whereupon Post responded by handing Stevens a lancet. Stevens declined by passing the lancet to Mott, who refused to receive it, and Post was allowed to exercise his discretion.

Post's last and most notable pioneer work was the ligation of the left subclavian, in its third part, for aneurism of the brachial artery. This was the eighth recorded ligation of the subclavian artery, the third which recovered, and the first in this country by the new method. The most interesting feature of the case was the rupture of the aneurism and the discharge of its contents during convalescence, with the final complete recovery of the patient.

From this review it appears that Dr. Post, previous to the year 1816, had applied the ligature successfully to five different arteries, twice to the carotid. His success has not been excelled, if we consider the complications he encountered, in any period anterior to antisepsis. The secret of his success, aside from

his great skill as an operator, is found in the extreme cleanliness, not only of his person, but of his instruments and the wound and dressings, thus securing asepsis. In the case of opening the peritoneum, he followed the operation with "an active cathartic, composed of an infusion of senna, manna, and cream of tartar, which caused frequent and copious discharges"—a form of treatment which some distinguished operators have latterly adopted as a preventive of peritonitis.

Brilliant as had been the career of Post in his pioneer work of introducing the new method of treating aneurism into American practice, Mott, his pupil, was destined to excel him in the number, variety, and severity of operations, and in the perfection and precision of details. He had a genius for scientific operative surgery. Nothing was done haphazard. Every detail, however minute and apparently unimportant, was carefully studied, and provision was made to meet every possible accident. He was by habit and training an aseptic surgeon. His personal neatness attracted public attention. His instruments were carefully cleaned before as well as after each operation, and every one assisting was required to be clean and to protect the wound and parts around it from every possible source of contamination. One of his pupils illustrated his habitual cleanliness as follows: Being present when another surgeon opened an abscess, Mott rolled up his coat sleeves, put his hands behind him during the operation, and, when the pus began to flow, proceeded to wash his hands as if he had been the operator.

Mott's training was well adapted to prepare him to take up the work which his preceptor was about to lay down. In addition to his pupilage under Post, he visited London and became an assistant of Mr. Astley Cooper. Cooper was the first to apply a ligature to the carotid artery for aneurism—1805—but unsuccessfully. In 1808 he repeated the operation, successfully, and Mott was present as his assistant and always spoke enthusiastically of this opportunity to witness what was considered pioneer work in operative surgery. A few months later, Cooper attempted to ligate the left subclavian between the scaleni muscles, but failed. Mott took part in this operation also, and was deeply impressed with the difficulties of the procedure and Cooper's skill and candor. He says: "After working indefatigably with all his eminent skill and superlative tact for an hour and a half, he abandoned the operation."

Mott's pioneer work began with the ligature of the arteria innominata. This was not only his greatest achievement in operative surgery, but it was the most brilliant operation ever undertaken by any surgeon in the history of operative surgery to that date. Nor has it ever been excelled in this department of surgery, if we give due weight to all of the circumstances attending the operation. It was by no means suddenly conceived and executed as an emergency operation, but was the ripe fruit of years of study and preparation. He states that "since the publication of Allan Burn's invaluable work on the surgical anatomy of the head and neck, I have been in the habit of showing, in my surgical lect-

ures, the practicability of securing, in a ligature, the arteria innominata; and I have had no hesitation in remarking that it was my opinion that this artery might be taken up for some condition of aneurisms, and that a surgeon with a steady hand and a correct knowledge of the parts would be justified in doing it." The proper case presented itself March 1st, 1818, and he says: "I could not for a moment hesitate in recommending and performing the operation." Dr. Wright Post, whom he had so often aided, now became his adviser and assistant.

Though the operation failed after giving the most encouraging prospect of success, Mott was not disheartened, but regarded its practicability and propriety as satisfactorily established by this case, and predicted that it would prove to be "the bearer of a message to surgery, containing new and important results."

The arteria innominata was repeatedly ligatured subsequently, but it was reserved for an American surgeon to secure the first successful result. The operator was Dr. A. W. Smyth, of New Orleans. The operation was performed in 1864. In this case the carotid was ligated at the same time, and on the fifty-fourth day the vertebral was also ligated.

Scarcely less memorable than Mott's operation on the arteria innominata, and creditable as a great surgical achievement, was Dr. J. Kearney Rodgers' ligation of the left subclavian, within the scaleni muscles. The operation was performed on the 14th day of October, 1845. It is an interesting fact that Mott was one of the consultants and opposed the operation, though he admitted that it might possibly "be tied by a careful and well-informed surgeon," yet he "considered that it was improper to do so." Colles, of Dublin, who was the first to ligate the right subclavian in its first part, condemned a similar operation on the left, stating that there was "such a combination of difficulties as must deter the most enterprising surgeon from undertaking this operation on the left side." Of the consultants, Drs. Mott and Stevens, though opposed to the operation, had such confidence in Dr. Rodgers's ability that they left the question of an operation to his discretion.

In his report of the case Dr. Rodgers says: "Although a decided majority of the consultation agreed as to the propriety of the operation of securing the artery for aneurism, still, as my colleagues kindly left it with me to decide whether it should be undertaken, I felt it incumbent on me to investigate the subject with great care, and accordingly gave it my most sedulous attention. I was the more anxious because, in the only case in which the attempt had been made by Sir Astley Cooper, in 1809, that eminent surgeon failed in securing the vessel. . . . I had always considered it as a perfectly justifiable operation, and one that a careful surgeon conversant with anatomy could accomplish if the tumor were of moderate size."

Rodgers did not hesitate to assume the responsibility which the action of his colleagues imposed upon him, and, true to his convictions of duty, proceeded to execute the trust committed to his care. The operation proved to be, in every

respect, as difficult as had been alleged, but he was fully prepared for every emergency. The ligature was successfully applied, and for several days everything promised success; but on the thirteenth day a hemorrhage occurred, which was repeated, and the patient died on the fifteenth day. The lesson which Rodgers drew from the operation was that the vertebral artery and, if possible, the thyroid axis should be secured at the same time by ligature. In concluding his report he says that, though the operation was unsuccessful in curing the aneurism, he trusts, "from the knowledge thence derived, we shall be enabled to enlarge our sphere of usefulness, and be the means of preserving human life."

John Kearney Rodgers (1793–1851) was born in this city in 1793. He was the son of Dr. J. R. B. Rodgers, an eminent physician of this city during the latter part of the last century. He was a graduate of Princeton College, New Jersey. He studied medicine under Prof. Wright Post, of the College of Physicians and Surgeons, and graduated in 1816. He then visited Europe and attended the lectures of Sir Astley Cooper, Brodie, Travers, and Abernethy. On his return he was appointed surgeon to the New York Hospital, on the resident staff of which he had served. He died of portal phlebitis, on the 9th of November, 1851, aged fifty-eight years.

Mott was the first surgeon who ligated the primitive iliac for aneurism. The operation was performed on the 15th day of March, 1827, and was executed with his usual care and attention to all of the details. The size of the tumor and the adhesions of the peritoneum rendered the procedure very difficult, but the operator was rewarded with the recovery of his patient, who was living thirty years after. In the practice of other surgeons the operation has proved very fatal. During the first twenty-five years after Mott's operation, the common iliac was ligated eighteen times with fourteen deaths—a mortality of upward of seventy-seven per cent.

The experience of Mott in the ligation of arteries was very great and his success far exceeded that of any contemporary surgeon. According to his own statement, he ligated the arteria innominata once, unsuccessfully; the common iliac once, successfully; the subclavian artery in its third part six times, all the cases were successful; the common carotid thirty-two times, with but five failures; the external iliac six times, with two failures (one patient died of drunkenness); the femoral fifty-three times, the failures being unknown. He had but one case of mortification of the extremity after ligature of an artery. This success Blackman attributed to Mott's great attention to the most minute details, both during the operation and during the subsequent treatment of his patients.

In 1812, Gibson, of Philadelphia, placed a ligature on a bleeding vessel in a gunshot wound of the groin, and after the death of the patient it was found that the injured vessel was the common iliac. The operation had no scientific value, and should not be classified with operations deliberately planned and executed.

The ligation of the external iliac was first performed in this country, as already

noted, by John Syng Dorsey. This was a most creditable performance, and antedated Post's operation three years. Dorsey operated August 19th, 1811. The special feature of his case was the use of an aneurismal needle, consisting of a blunt bodkin of silver, properly bent, and held in a curved forceps, the handles of which were firmly tied together. The curved forceps used on this occasion to pass the aneurismal needle was the invention of Physick, and was also used by Post in his operations. It is interesting to notice that a thermometer was employed to test the temperature of the limb, and it was found to become five degrees colder than the other.

Gratifying as was the success of American surgeons in their pioneer work in the ligature of arteries, and accurate as was the technique of the operation which they had devised, there was still a fatal defect which was to be remedied, viz., hemorrhage on the separation of the ligature. The practice of applying a silk ligature so tightly as to divide the inner coat of the artery, for the purpose of securing the union of the ruptured surfaces, was the rule with surgeons. The result was the gradual division of the artery by a process of ulceration due to the irritation of the unabsorbable ligature, and if union had not taken place, as too often happened, hemorrhage was the result. Physick, trained in the school of Hunter, suggested the remedy for this evil, viz., the use of "dissoluble" ligatures, the pressure of the internal surfaces of the artery together without injuring its coats, and healing the wound by first intention. At his suggestion, and under his directions, a series of experiments were performed with animal material, and French kid, which was absorbed after several days without injury to the artery, was selected, as described by Dorsey.

A very important contribution to the subject of animal ligatures was made in 1827 by Dr. Horatio Gates Jameson, of Baltimore, Md., in a prize essay, entitled, "Observations upon Traumatic Hemorrhage, Illustrated by Experiments upon Living Animals."

Horatio Gates Jameson (1778–1855) was born at York, Pa., in 1778, and graduated in medicine from the University of Maryland in 1813. He located in Baltimore, Md., and attained a high rank as a surgeon. He was the founder of Washington Medical College and professor of surgery, 1827–35. He died August 24th, 1855.

Jameson's conclusions were as follows:

1. If an artery is sufficiently healthy to admit of its obliteration by adhesion of its sides, it is best done by a ligature which will neither cut its coats nor strangulate, except in parts, the true vasa vasorum, so that the continuity of the vessel shall not be destroyed, although we obliterate its calibre.

2. If an animal ligature of the proper kind be properly applied, the vessel will be obliterated, the wound may be healed by the first intention, and the ligature will not cause suppurative inflammation, but in due time, being dissoluble, the whole will be removed by the absorbents; there will be no breach of con-

tinuity in the artery. . . . The vessel, which during the state of inflammation and effusion of lymph was converted into a cord, will pretty soon afterward be resolved into a flat string of white cellular structure.

The experiments of Dorsey and Jameson brought the operation of ligating arteries to scientific perfection by preventing secondary hemorrhage and securing healing of the wound by first intention.

An important feature of Jameson's experiments, to which he seems to have attached little importance, was the discovery that, as the animal ligature underwent absorption, it became "completely enveloped in a strong membranous capsule. . . . This arrangement of the capsule seemed to have the effect of drawing the button-like knobs (ends of the ligature, in the state of yellow pulp) together, and was thus closing the vessel. . . . The capsule covering the knobs or ends of the string was fully equal in strength to the outer coat of the artery, and therefore there was no tendency to hemorrhage." In the demonstration of this encircling ring or capsule which forms when animal ligature is employed for ligature, Jameson anticipated Lister, who describes it in his experiment as a ring of new tissue enveloping the dissolving animal ligature. He regarded it as of great importance in the prevention of hemorrhage. It certainly strengthens the artery at the point of ligature, where the artery has been rendered very weak by the strangulation of nutrient vessels. It is in effect like the provisional callus which forms at the seat of fracture of a bone—a temporary means of protecting a weak point in the vessel until repair takes place.

Jameson not only demonstrated at that early period the true method of procedure to secure success in the ligature of arteries by experiments on animals, but by a large series of operations in practice, as in ligating the carotid, the iliac, the femoral, the radial, and other arteries. But the real value of his teaching, though sustained by the authority of Physick, was not appreciated by contemporary surgeons. It was not until the introduction of antiseptics had awakened a new interest in measures for the prevention of suppuration of wounds that the practice of Jameson received the attention which it merited. Meantime his demonstrations had been forgotten, and the new method became popular as one of the features of antiseptic surgery. Essentially, however, the practice of to-day is along the lines laid down by Jameson. The employment of metallic ligatures, as silver wire, by Dr. Warren Stone, of New Orleans, and lead and other metals, has not proved as useful as animal material, owing to its liability to find its way out in time, with suppuration.

Amputation of limbs, which had been the subject of contention during the latter half of the seventeenth and the whole of the eighteenth centuries, was at its culmination on the opening of the nineteenth century. The questions at issue were: (1) Shall the method be the circular or flap, with their many modifications? and (2) shall the healing be by first intention or by granulation? In

1816 there appeared in Baltimore "*A Tract on Amputation*," by Prof. John B. Davidge, of the University of Maryland, the object of which was to introduce the "American method" of amputation. The "tract" is a very complete treatise on amputation, being a careful and critical review of the methods of procedure since the time of Celsus. The author states that he had been investigating the opinions and works of the surgical writers of France and Britain for ten or fifteen years, anxious to bring amputation to some degree of perfection. The "American method" is as follows: Two lateral semi-elliptical flaps are made, one on either side of the limb, consisting only of the skin and cellular tissue. These are dissected from the muscles and of a size sufficient to cover, freely and easily, the whole stump when laid together; that is, each flap must be at least the semi-diameter of the limb and so full as not to be in any way upon the stretch when laid down. The flaps being thus dissected from the muscles and reflected back, a circular cut is to be made with a large knife perpendicularly down to the bone, and completely around it; the muscles are now separated from the bone an inch or more farther up, and then, with the muscles well retracted, the saw is applied as closely as possible to the edge of the muscles, and the bone sawed off. The vessels being all well secured, the flaps are well coaptated and adjusted to the face of the stump, and maintained in position by adhesive straps, the ligatures being brought out of the lower angle of the wound.

The advantages claimed for this method are: 1. Complete drainage, thus preventing suppuration from retained fluids, as occurs when the wound is transverse. 2. The freshly cut surfaces are accurately applied to each other, which favors union by first intention, and no foreign body, except the ligatures, will "provoke inflammation or disquiet the economy of the parts." 3. The stump is more serviceable for future use than those left after other methods.

"The American method" was approved by many surgeons and was frequently performed with marked success, but it did not receive the attention which its merits deserved. Several years since, the writer made a careful study of the results of the different methods of amputation in our hospitals, and, coming to the same conclusion as Professor Davidge, drew similar figures of the line of incisions and the resulting stump, though unaware of the existence of this "Tract on Amputation."

John Beale Davidge (1768–1829) was born at Annapolis, Md., 1768. He attended medical lectures at Philadelphia and Edinburgh, but received his degree at Glasgow, 1793. He located at Baltimore, 1796. In 1807 he founded the Medical Department of the University of Maryland, and was the professor of anatomy and surgery, 1807–29. He took a prominent position as a surgeon, had a "pleasing address, very remarkable colloquial powers, and high professional character." He died in 1829.

The *reduction of dislocations* was a subject of great interest to British surgeons. According to Mr. Pott, the leading authority in British surgery during

the latter half of the eighteenth century, dislocations were reduced by force. Of the machines for that purpose, he says: "Many or most of them are much more calculated to pull a man's joints asunder than to set them to rights." With true scientific intuition he declares that "replacing a dislocation would require very little trouble or force, were it not for the resistance of the muscles and tendons attached to and connected with them." Little if any useful progress was made in the direction pointed out by Pott, to determine the principles governing the reduction of dislocations, until the attention of American surgeons was directed to the subject. Now, the most formidable of these dislocations, those of the femur at the hip-joint, are reduced in American practice without violence or pain, by simple manipulation of the limb with the hands. The several steps in the process of investigation, by which the principles governing the natural and rational method of reducing all dislocations was discovered, illustrate the scientific spirit of American practitioners as well as teachers of surgery:—

Occasional reductions of dislocations at the hip-joint during manipulations of the limb, even after protracted efforts had been made at reduction with powerful machines, have been recorded from the time of Hippocrates. But they were regarded as accidents, having no scientific value. Physick reduced a dislocation of the femur by manipulation in 1812, after the pulleys had failed. He believed that the cause of previous failure was due to the escape of the head of the bone through a rent in the capsule, and that the head had become fixed as in a button-hole, from which he dislodged it.

In 1831 Dr. Nathan R. Smith, of Baltimore, Md., published "*Remarks on Dislocations of the Hip-joint*," and states that the principles which he endeavors to establish, relative to reduction, were derived in part from the lectures of his father, Dr. Nathan Smith, professor of surgery in Yale College. It appears from the record that as early as 1811 his father explained a method of reducing the dislocations of the femur at the hip-joint by manipulations of the limb with the hands, without the aid of mechanical appliances. His method of procedure was based on a careful study of the action of the muscles attached to the upper extremity of the femur. The author thus states his conclusions: "There is, no doubt, a constant mechanical principle upon which the reduction is effected in such cases, and one which would perhaps succeed in nearly all cases if we knew how to employ it understandingly and with precision, and did not avail ourselves of it by mere haphazard. If a gentle movement of a peculiar kind succeed in one case of complete dislocation on the dorsum ilii after all other means have failed, ought not this movement, if well understood, to succeed in other cases better than the usual mode? The mechanism of these dislocations is certainly the same in all of this variety, . . . furnishing the same impediments and the same aids in every case. This frequent failure of art and the success of accident satisfy me that there is some important principle relative to the mechanism of these dislocations which is not understood. Accident ought not to accomplish

the reduction of a bone with more ease than art. When it does so, such accident should be our instructor, and teach us the mechanism by which it operated, and this we should repeat in similar cases."

The author then proceeds to discuss the mode of applying force in the reduction of dislocations of the hip, and, in illustration of the adaptation of manipulation to meet the varying action of the muscles affected by the dislocation as compared with the pulleys, he mentions the case of reduction by Physick and adds a case narrated by his father, "in which he promptly succeeded by the mere force of hands in effecting reduction." The description of the muscles concerned in reduction is given in detail and the method of manipulating the limb is illustrated with engravings. "The free flexion of the thigh upon the pelvis" was regarded "as a very important part of the compound movement." For twenty years no further attention seems to have been given to the subject, and it is doubtful whether the views of Nathan Smith had become known to the profession at large.

In 1851 Dr. William W. Reid, of Rochester, N. Y., published a paper on "*Dislocation of the Femur on the Dorsum Ilii. Reduction without Pulleys or Any Other Mechanical Power.*" He states that he had been present at several operations for the reduction of dislocations at the hip-joint by means of pulleys, and was impressed with the apparently unnecessary force employed. For ten years he studied the mechanism of these dislocations, and came to the conclusion that "the difficulty lay in the *extension* of the . . . adductors and rotators, and that all traction . . . on the dislocated bone only increased this tension, and could do nothing toward bringing it into place, except at the hazard of almost certain rupture of some of these muscles or of fracture of the neck."

Guided by this conclusion and the experience gained by his experiments, Reid practised manipulations and evolutions on the skeleton until he had determined that "dislocation of the femur on the dorsum ilii . . . is reduced with the greatest ease in a few seconds or minutes, without much pain, without an assistant, without pulleys . . . or any other mechanical means, simply by flexing the leg on the thigh, carrying the thigh over the sound one upward over the pelvis, as high as the umbilicus, and then by abducting and rotating it."

Reid gave wide publicity to his paper and discussed his method before medical societies. Surgeons in hospital practice who tested its merits found that it was a vast improvement upon the pulley and other appliances.

In 1853 Dr. Moses Gunn, professor of surgery in Rush Medical College, Chicago, who had taught Reid's method, published an account of experiments made in 1851–52 to determine the obstacles to reduction of hip-joint dislocations. His conclusion was that the "untorn portion of the capsular ligament, by binding down the head of the dislocated bone, prevents its ready return over the edge of the cavity to its place in the socket." He rejected the opinion of Reid that the opposing forces are the muscles. The untorn portion of the capsular

ligament to which he refers is "the *anterior and inferior* half of the capsule" (which includes the ilio-femoral or Y-ligament of Bigelow), and this, he states, is "the *sole agent* which gives character to those dislocations, and, with the exception of the fascia lata, *the only obstacle to be overcome by our efforts to reduce them.*"

In 1861 Dr. Henry J. Bigelow, professor of surgery in the Harvard Medical College, who also had taught Reid's method, was led to expose a joint, the luxation of which had been the subject of a lecture, and was surprised to observe the simple action of the ligament (the anterior and inferior half of the capsule, alluded to by Gunn). The dislocated joint was in the following condition: 1. Great laceration of the muscles about the joint. 2. The ligamentum teres broken. 3. Laceration of the inner, outer, and lower parts of the capsule. 4. The anterior and upper parts of the capsule uninjured, and presenting a strong fibrous band, fan-shaped, and slightly forked. On dividing the remaining tendinous and muscular fibres about the joint, excepting this fibrous band, it was found that the four commonly described dislocations of the hip could still be exhibited without difficulty, and that in each of them the anterior portion of the capsular ligament, which alone remained, sufficed at once to direct the limb to its appropriate position and to fix it there. On the other hand, if the entire capsule of the hip-joint be divided and the muscles left intact, these dislocations are but imperfectly represented.

The conclusion of Professor Bigelow, as a result of his investigations, was that the muscles play but a subordinate and occasional part, either in hindering reduction or in determining the character of the deformity, but that these conditions are chiefly due to the resistance of the ligament. The practical result of this conclusion is thus stated by Bigelow: "The theory here advanced recognizes the anterior portion of the capsular ligament as the exponent of the total agency of the capsule in giving position to the dislocated limb, and, what is more important, is so identified with the phenomena of luxation that reduction must be accomplished almost wholly with reference to it."

This discovery led Bigelow to review the whole subject of dislocations at the hip-joint and determine the peculiarities of each and the special methods of reduction applicable to the different forms. The course of study which he pursued in demonstrating the Y-ligament and its relations to the position of the head of the femur in the several dislocations, and the exact direction in which the forces employed in reduction should be applied, forms one of the brightest chapters in scientific surgery.

Thus, after a generation of investigation by American surgeons, the "constant mechanical principle upon which the reduction is effected" in hip-joint dislocations, suggested by Nathan R. Smith, was discovered, and the method of Reid, by which a dislocation on the dorsum ilii "is reduced with the greatest ease in a few seconds or minutes, without much pain, without an assistant, without

pulleys," was made applicable to all dislocations at the hip-joint. Though Bigelow gives the paternity of the new method, which is concisely expressed in the words "flex, abduct, evert," to Nathan Smith, the world is indebted to Reid for the practice of reducing dislocations on the dorsum ilii by manipulation, and to Bigelow for an extension of that method to all other forms of dislocations at the hip-joint.

The *treatment of fractures* was vigorously discussed by foreign surgeons at the beginning of the last century. Samuel Cooper, author of the "*Surgical Dictionary,*" and historian of British surgery, referring to the comparison of French and English surgery by Roux, 1814, states that, with the exception of the teachings of Pott, "it cannot be said that we had made a single improvement of consequence in the treatment of any particular fracture."

We shall illustrate our subject by noticing the improvements made by American surgeons in regard to a single fracture, viz., that of the femur. Fractures of this bone were the theme of constant discussion by French and British surgeons at the close of the eighteenth and the beginning of the nineteenth centuries. The controversy had become somewhat of a national issue. Pott, on the part of the British surgeons, advised that the limb, flexed at the hip and knee, be laid on its side, supported only by lateral splints loosely applied, the body being inclined to that side. His contention was that, by thus relaxing the muscles, the fragments fall into position and require no other support than side splints. Desault, on the part of the French, placed the limb in an extended position and applied an external splint from the crest of the ilium to a point below the foot and attempted extension and counter-extension as the governing principle in the treatment of these fractures.

Though both of these methods had merits, in practice they proved defective. The relaxation of the muscles effected by Pott's method was very desirable, but it was impossible to maintain the limb quiet in that position without more restraint from appliances than the two splints supplied. The extension and counter-extension by means of a long side splint in Desault's method was very important, but the plan of securing it was inefficient; the extending and counter-extending bands, acting obliquely, tended to draw the upper fragment and the foot outward, while that at the foot caused painful excoriations at the ankle. The two principles on which these methods of practice were based were not, therefore, effectively applied by the means devised by their respective advocates.

American surgeons took an active interest in the treatment of fractures of the femur, and at an early period began to make improvements upon the French and British methods of practice. Physick, if we accept Dorsey as authority, preferred Desault's straight position, for the reason that "the muscles very speedily accommodate themselves to the new position caused by the action of the extending and counter-extending bands at the extremities of the splint."

As the external or long splint extended only from the crest of the ilium to a point beyond the sole of the foot, the upper band around the thigh, acting obliquely to the shaft of the femur, tended to draw the upper fragment outward and thus prevent coaptation of the fractured surfaces; while the lower band, acting in a similar manner, turned the foot outward and displaced the lower fragment. To remedy this defect Physick extended the splint upward to the axilla and placed a crutch-like form on the end to prevent rubbing against the patient's side. The perineal extending band, fastened to the upper end of the splint, now made traction nearly in line with the shaft of the femur, and no irritation of the hip occurred. This improvement was made about 1800.

To remedy the defective action of the band at the foot, he added a transverse foot-piece, over the end of which the extending band was passed, which made traction in line with the shaft of the femur. These improvements greatly increased the efficiency of the splint and added to the popularity of the treatment in the straight position. Dorsey says: "I have for twelve or fourteen years witnessed the effect of this mode of treatment in the Pennsylvania Hospital, where more accidents are admitted than in any other institution in America, and I am safe in asserting that the success of the practice has been surpassed by that of no other hospital in the world." He admits that some surgeons have become dissatisfied with this mode of treatment, owing to the excoriations caused by the bands especially on the foot.

It was in this particular feature of extension and counter-extension that American surgeons perfected the method of treatment of fractures of the femur in the straight position. In 1861 Dr. Gurdon Buck, of New York, published an account of the method of treating fractures of the thigh in the New York Hospital, with illustrations. This was the most important contribution that had to this time been made, as it remedied the great defects of the methods hitherto employed to effect extension. "Buck's extension" is too well known to the students of surgery to require explanation. It is sufficient to state that its publication was the culmination of a century of persistent effort on the part of the most reputable surgeons of this country and Europe.

Fig. 21.—Gurdon Buck (1807–1877).

Gurdon Buck (1807–77) was born in New York City, May 4th, 1807. He graduated in medicine from the College of Physicians and Surgeons, New York, in 1830, and entered the New York Hospital as a member of the resident staff, serving by preference on the medical division. In 1833 he went abroad and spent two years in the hospitals of Paris, Berlin, and Vienna. On his return he located in New York, and in 1837 was appointed one of the visiting sur-

geons of the New York Hospital, a position which he held until his death, a period of forty years. During his connection with this hospital it received most of the surgical cases of the city, and its surgical staff was constituted of the most eminent surgeons of New York. He was appointed surgeon of St. Luke's Hospital on its organization, and subsequently occupied the same position in the Presbyterian Hospital. Dr. Buck was a most painstaking and successful practitioner and made improvements in many branches of surgery, as in the operation for bony ankylosis at the knee-joint, in the treatment of œdema glottidis, and in lithotomy and lithotrity. He was also greatly interested in plastic surgery, and published, in 1876, a monograph entitled "*Contributions to Reparative Surgery.*" But the apparatus which he devised for the application of traction in the treatment of fractures of the femur will long be regarded as his most useful contribution to the practice of surgery. He died March 6th, 1877.

Lithotomy was an operation of the first importance during the eighteenth century, and the various methods of procedure were subjects of endless discussion. American surgeons who were educated abroad returned to practise in this country, ambitious to gain reputation as successful lithotomists. Many became good operators and some attained an eminence equal to that of the most reputable lithotomists abroad. Dr. John Jones, of New York, had a wide reputation as a successful lithotomist as early as 1760. Physick, of Philadelphia, was not only a successful operator, but he improved the gorget. Jameson, of Baltimore, was a skilful operator and advocated healing the wound by first intention by placing the patient on his side, with a catheter retained in the bladder. Dr. Benjamin W. Dudley, of Kentucky, became famous for the large number of operations which he performed, amounting to two hundred and twenty-five, and for his great success, having one hundred consecutive cases without a death. But in the matter of numbers, Dr. John P. Mettauer, of Virginia, excelled him, having a record of four hundred cases.

In 1824–25, according to Jameson, of Baltimore, some of the most distinguished surgeons of America performed Civiale's operation of "lithontrity" as a substitute for lithotomy, "in all which attempts there were complete failures." The operation was, however, subsequently advocated by Randolph, Gibson, Nathan R. Smith, and others, under the title "lithotripsy," but it did not supplant the older operation of lithotomy.

In the progress of American practice both lithotomy and lithotripsy were destined to be supplanted. Bigelow, impressed with the great distensibility of the urethra as shown by Otis's experiments, began to use much larger evacuating tubes in the operation of lithotrity, with the result of being able to remove much larger fragments of the stone than formerly, and of thus reducing both the number of operations and the length of time of each trial. The new operation was gradually perfected under the title of "litholapaxy" (evacuation), and it has largely superseded all other methods of removing calculi from the urinary bladder.

"*Hip-joint disease*" was a fatal or a crippling affection of childhood which surgeons regarded as helpless and hopeless by any method of treatment known prior to the year 1800. But American surgeons have stricken hip-joint affections from the category of incurable diseases and placed it among the more simple and curable forms of sickness peculiar to childhood. The method of treatment was determined after careful study of the pathological conditions of the hip-joint by three surgeons at different periods. The history of their work admirably illustrates the process of evolution of practice based on scientific principles.

Dr. Physick returned from Europe a student of scientific surgery as taught by his preceptor, Hunter. One of these principles was that *rest* is the first and essential factor in the correct treatment of inflammation. He applied that principle to the treatment of hip-joint disease, about the year 1800, in the following manner: He employed "a splint properly carved so as to be adapted to the irregular size, shape, and position of the diseased hip-joint, thigh, and leg. It must also be carved so as to fit the principal part of the same side of the trunk. The whole must be long enough to extend nearly half way round the parts to which it is applied. In those cases in which the thigh is bent upon the pelvis and the leg upon the thigh at the knee joint, the surgeon must by no means attempt to force the limb into a straight splint. . . . The splint must be made angular at those parts so as to adapt itself to the exact position of the limb, however crooked it may be. After the patient has worn a splint of this shape for some time, the inflammation and swelling become so much relieved that the limb can be placed in a much straighter position; and now it becomes necessary to have a second splint constructed, which will adapt itself to the altered condition of the parts."

Randolph, Physick's son-in-law, makes the following statement of the results of the new method of treatment which imperfectly secured *rest* to the inflamed joint: "The success which Professor Physick has met with from his mode of treating hip-joint disease has been so highly encouraging as to induce him to believe that he can effect a cure in all recent cases, and many even of long standing, provided the joint be not disorganized."

The next step of progress was the cure of the "disorganized" cases. In 1853 Dr. Alden March, of Albany, N. Y., published the results of a series of studies in the pathological museums of this country and Europe, for the purpose of determining the condition of the hip joint in advanced disease. His conclusion was that the pressure of the two inflamed joint surfaces led to destructive ulceration of the cartilages, and his practical inference was that if traction of the limb were added to fixation of the joint, sufficient to relieve the pressure of the head upon the surface of the acetabulum, not only would ulceration be prevented, but cases where ulceration already existed might be cured. To meet this indication he applied the long splint, as in fracture of the thigh, with extension and counter-extension. The result was immediate relief to the pain and final recovery of

the more intractable cases. Greatly as this method improved the treatment of Physick and enlarged the number of curable cases, there was still a class of feeble patients who could not bear the long confinement and large suppuration that followed the necrosis of the head of the femur, and eventually succumbed to exhaustion.

The final question in the problem of treating hip-joint disease was, therefore, How can fixation of the joint and traction of the leg be effected while the patient is allowed to walk? The clew to the answer was given by Dr. Henry G. Davis, a surgeon of great inventive skill, of Worcester, Mass., in 1860. He devised a splint which imperfectly effected the object, but which suggested to those engaged in that special field of work the proper apparatus. Davis's splint especially impressed Dr. Lewis A. Sayre, of New York, who was devoting much attention to this disease, and who states that he had long recognized the importance of an appliance that would secure fixation, traction, and ability to walk, and endeavored to construct such an apparatus, but did not succeed. On examining Davis's splint, Sayre readily discovered not only the essential features of a rightly constructed hip splint, but he detected the real cause of the failure of the inventor to meet conditions necessary to success. He immediately undertook to construct a splint which would meet the indications now so apparent to him, and the result was a splint which has since been known by his name and which is the perfection of surgical art. As a teacher and author, Sayre so clearly and persistently demonstrated and illustrated the scientific treatment of hip-joint disease that he compelled the profession to adopt the new method.

The result of this half-century of studies relating to hip-joint disease is in the highest degree creditable to American surgeons. The class of children that at the beginning of this period died after years of intense suffering, confined to their beds, are to-day met on the streets, at school, and on the playgrounds, in the enjoyment of healthy activity. A death from hip-joint disease is unknown in mortuary statistics.

Orthopedic surgery, as a specialty, had its origin in this country in the clinical lectures of Dr. William Detmold (1808–1900), of New York. He was a native of Hanover, Germany, a graduate of the University of Gœttingen, and a pupil of the famous orthopedist, Stromeyer. He located in New York City in 1837, and devoted himself to the practice of orthopedic surgery. For the purpose of giving instruction in this specialty he established a public clinic, and gave courses of lectures which were largely attended by medical students and practitioners. He was a skilful operator and introduced Stromeyer's method of tenotomy, which he practised with the greatest freedom. Though Detmold discontinued his clinics prior to 1860 and rarely published papers, it was through the influence of his teaching that the first impulse was given to an interest in orthopedia, which resulted finally in raising it to the position of a distinct branch of surgical practice.

Detmold as a teacher of orthopedia was succeeded by Dr. Lewis A. Sayre, then an enthusiastic young surgeon who was devoting himself to the treatment of cases of deformity. As one of the founders of the Bellevue Hospital Medical College, 1861, he urged the establishment of a professorship of orthopedia, and on its creation he was appointed to the position, the first in this country. Sayre's annual course of lectures gave a powerful impulse to the study and practice of orthopedic surgery throughout the entire country. His attractive personality, his resistless enthusiasm, his vast resources for clinical illustrations afforded by Bellevue Hospital, and his bold and often brilliant operations, inspired students and practitioners, gathered from all parts of the United States, with a genuine determination to practise orthopedia. The influence of Sayre's teaching was greatly increased by the publication of his lectures in 1876, which formed a complete treatise on orthopedic practice. Though many surgeons occasionally performed operations for deformities, and frequently papers were published narrating individual cases and methods of treatment by apparatus, it was not until the Bellevue school had created a professorship of orthopedia, and Sayre had given to that professorship the character and importance of a special branch of surgical education, that orthopedia assumed the position of a specialty in surgical practice in this country.

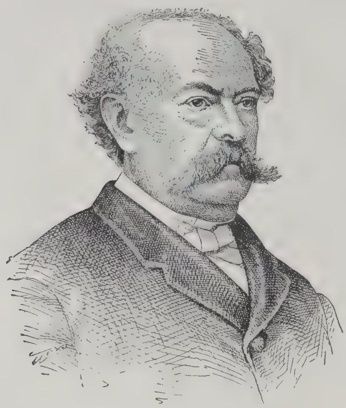

Fig. 22.—William Detmold (1808–1900).

Lewis Albert Sayre (1829–1900) was a native of New Jersey and a graduate of Transylvania University, Kentucky. He studied medicine in New York City and graduated from the College of Physicians and Surgeons in 1842. In the same year he was appointed prosector to the chair of surgery in that institution, a position which he held until 1852. In 1853 he was appointed one of the visiting surgeons of Bellevue Hospital. In 1861 he was one of the founders of Bellevue Hospital Medical College, and at his suggestion the professorship of orthopedia was established, which was subsequently assigned to him. He retained this position until 1897, when ill health compelled him to retire. He died in 1900.

Fig. 23.—Lewis Albert Sayre (1829–1900).

Gynæcology as a special branch of operative surgery had its origin in the experimental work of Dr. J. Marion Sims. "Silver as a suture is the great surgical achievement of the nineteenth century," was the declaration of this pioneer

surgeon in his anniversary discourse before the New York Academy of Medicine in 1857. In this discourse Sims describes at length and eloquently two of the most important events in the history of the American practice of surgery, viz., the introduction of silver wire as a suture, and the method of curing vesico-vaginal fistula. The two discoveries were the result of a single course of experimental studies, "conducted," as the author states, "on the principles of a rational inductive philosophy." The original purpose and object of Sims was the cure of vesico-vaginal fistula. After repeated failures and a careful study of everything connected with the operation that might contribute to his want of success, he was finally, after four years of patient effort, led to the conclusion that the silk suture was the cause of failure.

Instead of abandoning his enterprise, he turned his attention to the solution of another problem, apparently more difficult than the one on which he had expended so much time and study. The question now to be settled was, What material can be used for suture that will not, like silk, act as a seton? He had read the experiments of Levert, of Mobile, Ala., made in 1829, at the suggestion of Physick, which proved that wire or lead caused no irritation, and also the statement of Mettauer, of Virginia, that he had used lead wire in operations with success. Sims had, in fact, used lead wire in his experiments, but without success, and therefore he turned to silver as offering more advantages than other metals. He operated with silver wire on the 21st of June, 1849, upward of three years after his first experimental operation, and with entire success.

The value of that operation in the relief of human suffering, by the powerful impulse which it gave to operative surgery, can never be estimated. Sims first published the details of the operation in 1852. It established the specialty of "gynæcology," of which Sims is the founder, but in the comprehensive scope of his work he was a general surgeon.

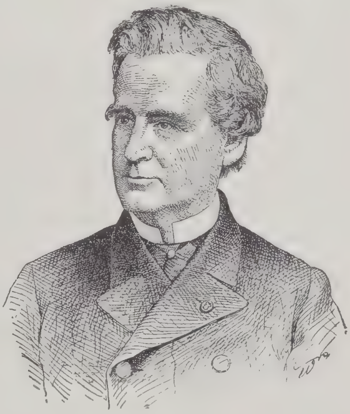

Fig. 24.—J. Marion Sims (1813–1883).

J. Marion Sims (1813–83) was born in South Carolina, January 25th, 1813. He graduated in medicine at Jefferson Medical College in 1835, and located in Montgomery, Ala. In 1853 he removed to New York, where he was successful in establishing the Woman's Hospital, in which he practised gynæcology, clinically, rendering popular his operation for the cure of vesico-vaginal fistula. He repeatedly visited the European capitals, where he performed his special operations, and received honors and decorations from the French, Italian, Spanish, and Belgian Governments. He invented many instruments adapted to the operations which he was accustomed to perform. He was in Paris at the breaking out of the Franco-Prussian War, and was made surgeon-in-chief of what was called the

"Anglo-American Ambulance Corps," composed of eight Americans and eight Englishmen. This organization was at the battle of Sedan and was assigned to duty in connection with a large hospital where upward of twenty-six hundred wounded were treated. Dr. Sims continued his connection with the Woman's Hospital from its organization in 1855 to his death in 1883.

The series of studies which have terminated in the scientific *treatment of diseases of the appendix vermiformis* enters largely into the American practice of surgery. The initial step in these studies was taken in 1856, on the publication of a paper by Dr. George Lewis, entitled, "*A Statistical Contribution to Our Knowledge of Abscess and Other Diseases Consequent upon Lodgment of Foreign Bodies in the Vermiform Appendix, with a Table of Forty Cases.*" Lewis was a young physician of New York who had recently had a fatal case of appendicitis, and at my suggestion made the collection of cases which formed the basis of his paper. Professor Kelly, of Baltimore, in his great work on "Appendicitis," speaks of Lewis's paper as "by far the most complete investigation of the diseases of the appendix up to the date of its publication."

In this paper Lewis brought prominently to the attention of American surgeons the operation of Hancock, of London, who in 1848 deliberately opened an abscess formed in the region of the appendix and as a result of its diseased condition, and cured his patient. Lewis's paper excited great interest among surgeons, and led Dr. Willard Parker, of New York, to repeat the operation of Hancock. In 1867 Parker published the histories of four cases on which he had operated, and in all but one he had not operated until fluctuation was distinct. In his early cases he feared to operate, as he was uncertain of the diagnosis, but in one case he ventured to operate in the early stage of the disease, and saved his patient. He did not, however, advocate an early operation, but advised a delay of five days, as a rule, in order that it might be determined whether suppuration had occurred, believing, as I often heard him remark, that an operation was required only when it was certain that pus had formed. Professor Kelly states of Parker's work: "From the date of his teaching operative treatment of appendicitis began an evolution which ended in the revolution of surgery."

But Parker's operation only sought the evacuation of pus, as in opening an ordinary abscess, the offending gangrenous appendix being left in the wound. The next step was the removal of the appendix, which was done in this country in May, 1886, at Roosevelt Hospital, New York, by Dr. R. J. Hall. This operation was only an incident in the case. The next step in advance was to be the excision of the appendix as a necessary part of the operation. Parker's operation was performed from time to time, and in 1875 Gouley tabulated twenty-five cases. There was a reduction of mortality from forty-seven per cent in 1867 to fifteen per cent in 1882. The removal of the appendix as a necessary part of the operation was not, however, undertaken until 1887, when Dr. Thomas G.

Morton, of Philadelphia, deliberately planned and executed its removal, thus perfecting Parker's operation.

Many questions still remained unsettled, especially as to the diagnosis, the cases requiring operation, the exact time of operating, and the method of procedure. These questions were finally very definitely settled by two remarkably able scientific papers. The author of the first was Dr. R. J. Fitz, of Boston, whose article, "*On Perforative Inflammation of the Vermiform Appendix,*" appeared in 1886; and so thoroughly were these doubtful questions discussed and determined that the paper has been pronounced an "epoch-making memoir." Professor Kelly says that Fitz "has done more than any single individual to bring about a right understanding of the morbid conditions affecting the vermiform appendix." Again he refers to Fitz's work as follows: "The time was ripe, the man appeared, and surgeons, needing but the assurance of safety, gratefully accepted this transfer from the domain of internal medicine, and began with alacrity to develop the operative procedure."

The paper of McBurney, published in 1889, was a critical review of all questions relating to the operation, and its conclusions determined the details of procedure with so much precision that there have been only minor changes in the methods which he prescribed. Thus, commencing with the investigations of Lewis and the tentative operations of Parker, and terminating with the scientific inductions of Fitz and McBurney, in their classical papers, have American surgeons established the proper treatment of inflammatory affections of the appendix vermiformis.

It has not been our purpose to notice the achievements of individual surgeons except as they have resulted in important reforms in practice. But there have been instances where surgeons have performed acts or adopted methods to meet conditions hitherto unknown to them, which illustrate American ingenuity and enterprise. Several of these examples deserve notice.

Amputation at the shoulder joint was introduced into practice during the eighteenth century by French surgeons. The first operation in this country was performed by Dr. John Warren, of Boston, as early as 1781. Dr. Warren had had a large experience in operative surgery during the Revolutionary War. The operation was performed in the Military Hospital at Boston, where Dr. Warren was giving lectures to physicians and students. The details of the operation were not published, but it was successful.

In 1792 Dr. Nathan Smith *trephined bone* for the cure of an abscess. The patient was aged nine years; there was a collection of matter in the thigh, extending from above the knee nearly to the trochanter. An incision was made from near the knee joint upward eight inches; a large discharge of pus took place, and the bone was found denuded of its periosteum two-thirds of its length. He determined to wait and see if granulations would appear on the denuded

bone; but as they did not, and the bone became of a dark color, he decided to remove a portion in such manner as to go through the dead part, let that be more or less. He used a trephine—"the round saw employed in operating on the skull"—nearly in the centre of the denuded part, and removed a piece of bone down to the medullary substance. Purulent matter issued in pulsations from between the bone and the medullary substance. In a few days "the bone, which was a pearly white, a little verging to brown, where exposed to the external air, changed its appearance, assuming a carmine color, and finally recovered, with no other loss of substance than a thin scale."

Previous to the year 1806, amputation at the hip joint had been performed but once by British surgeons, and in that case the operation resulted fatally. In that year Dr. Walter Brashear, of Kentucky, performed this amputation successfully. The operation consisted of two procedures: First, the surgeon amputated at the middle third of the thigh in the usual way and ligated the vessels; second, he made an incision on the outside of the limb from the point of previous operation to the hip joint. Then he detached the soft parts from the bone and disarticulated it. The patient made a good recovery.

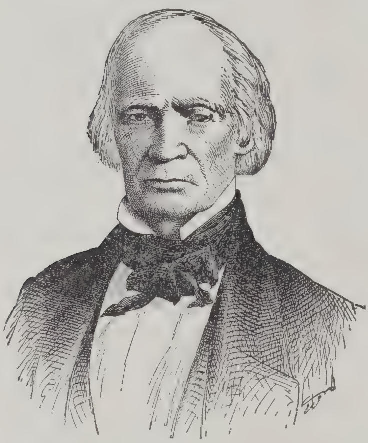
FIG. 25.—Walter Brashear (1776–1809).

Walter Brashear (1776–1809) was a native of the State of Maryland. He received his education at the Transylvania University, Kentucky. He attended a course of lectures at the University of Pennsylvania and then travelled extensively; on his return he engaged in merchandise for twelve years, when he resumed practice at Bardstown, where he performed the amputation. He removed soon after to Lexington, Ky., and after a few years of successful practice he retired to the State of Louisiana. He was not a graduate in medicine. He died in 1809.

The first applications of a ligature to the common carotid were for the arrest of hemorrhage in open wounds. In the performance of this operation American surgeons were anticipated eighteen days by British surgeons. On the 4th of November, 1803, Dr. Mason Fitch Cogswell, of Hartford, Conn., attempted to remove a tumor which developed in the parotid gland and parts adjacent; in the progress of the dissection the tumor had to be separated from the carotid artery, which it surrounded. The effort failed, and the operator placed a ligature around the artery, which he then severed. The case progressed favorably, the ligature separating on the fourteenth day, but on the twentieth day one of the anastomosing arteries under the forepart of the jaw began to bleed, and, no effort being made to check it for a considerable period, the loss of blood was so great that the patient sank and died.

The operation of Dr. Cogswell was entirely original with him, as was the

second case, by Dr. Amos Twitchell, of Keene, N. H., original with that surgeon. This case was one of sloughing of the internal carotid following a gunshot wound; the patient made a good recovery. This operation was performed on the eighteenth day of October, 1807.

The first case of ovariotomy, by Dr. Ephraim McDowell, of Kentucky (1771–1830), was deliberately planned and executed by a surgeon who had never "seen so large a substance extracted, nor heard of an attempt or success attending any operation, as this required." The woman rode sixty miles on horseback to the place of operation. The operation was performed in December, 1809, by an "incision about three inches from the musculus rectus abdominis, on the left side, continuing the same nine inches in length, parallel with the fibres of the above-named muscle, extending into the cavity of the abdomen, the parietes of which were a good deal contused, which we ascribed to the resting of the tumor on the horn of the saddle during her journey. The tumor then appeared full in view, but was so large that we could not take it away entire. We put a strong ligature around the Fallopian tube near the uterus; we then cut open the tumor, which was the ovarium and fimbrious part of the Fallopian tube, very much enlarged. We took out fifteen pounds of a dirty, gelatinous-looking substance, after which we cut through the Fallopian tube and extracted the sac, which weighed seven and a half pounds." The wound was closed with interrupted sutures and adhesive strips between them, and the ligature on the Fallopian tube was brought out of the lower angle of the wound. The report adds: "In five days I visited her, and, much to my astonishment, found her engaged in making up her bed." The patient returned home in twenty-five days in good health.

FIG. 26.—Ephraim McDowell (1771–1830).

It is reported that the operation created such public opposition that a mob collected around the house in which it was performed, prepared to attack the surgeon if he failed. An account of the operation was published several years after in an obscure journal, and was so imperfectly reported as to be discredited; hence it has had no other importance than an historical incident.

Twelve years later, in 1821, Dr. Nathan Smith, of New Haven, Conn., performed the operation of ovariotomy, having no knowledge of any previous similar operation. He was led to make the operation from his observations in dissecting the body of a patient who had died of ovarian dropsy after being tapped seven times. The sac was found to be the right ovarium, which filled the whole abdomen, but it adhered to no part except the proper ligament, which was no

larger than the finger of a man. He had seen two other autopsies of women who suffered from ovarian disease, and noticed that the sacs were unattached, except to their own proper ligaments. He inferred that while the tumor remained movable it might be removed with a prospect of success. His operation was as precise in all its details as the most modern method. The external incision began about an inch below the umbilicus, directly in the linea alba, and extended downward three inches; the sac was evacuated with trocar and cannula and then drawn out, bringing with it a considerable portion of omentum, which was separated and the bleeding vessels tied with leather ligatures. When the ovarian ligament was brought out it was cut off, two small arteries were tied with leather ligatures, and the stump was returned; some adhesions of the sac were separated and the mass was removed. The incision was closed with adhesive plaster and a bandage applied over the abdomen. No unfavorable symptoms occurred, and in three weeks the patient was able to walk about.

Smith's well-devised and executed operation had no proper publicity, and hence it had no effect in introducing a new procedure into practice, but, like McDowell's operation, simply illustrates the great abilities of the individual surgeon.

Surgery of the abdominal cavity began to attract attention toward the close of this period, but the few operations that were practised were incidental and accidental rather than deliberative. There had been some preparatory work done, as in the experiments on the treatment of wounds of the intestines. As early as 1805 Dr. Thomas Smith published a thesis presented to the faculty of the Medical Department of the University of Pennsylvania, entitled, "*On Wounds of the Intestines.*" The thesis was based on the results of twelve experiments on dogs, undertaken to prove the value of the different methods of treatment of wounds of the intestines. He made transverse and longitudinal wounds, divided the tube, exsected portions, and cut away triangular sections. He used the interrupted and the continuous suture. The vivisections were very carefully made and the results accurately stated. This paper was highly creditable as an effort to determine, at that early day, by scientific inquiries, the proper method of treating wounds of the intestines. He used the silk suture, and found that when he cut the thread near the knot, returned the bowel, and permanently closed the external wound, he had better results than in cases where he followed the common practice of allowing the ends of the suture to depend from the wound for the purpose of removal when it separated. Contrary to the prevailing views, he found that longitudinal wounds healed as promptly as transverse wounds.

Dr. S. D. Gross, then professor of surgery in the University of Louisville, Ky., published (1843) his monograph, entitled, "*An Experimental and Critical Inquiry Into the Nature and Treatment of Wounds of the Intestines.*" The object of the author was to "inquire into the process employed by nature in repairing wounds of the intestines," and "particularly to determine, if possible,

the value of the more important methods of treatment recommended from the time of Ramdohr down to our own."

But the great operations of ovariotomy by Dudley and Nathan Smith stood as permanent beacon lights for half a century, indicating the direction of the explorer for new fields of conquest, before the pioneer appeared who dared to penetrate the peritoneum and effectively treat the viscera which it invested. Dr. J. Marion Sims, guided by the same inductive method of reasoning and inspired by the scientific spirit which characterized his introduction of silver wire into practice, not only advocated the free exposure of the peritoneal cavity for the purposes of surgical operations, but he boldly led the way in his operation for gall stones. The result of his pioneer work has been the almost limitless expansion of the field of operative surgery.

Though anæsthesia was introduced into the practice of surgery in 1846, and exerted a marked influence upon its evolution during a quarter of a century of the period of which we write, it was not until antisepsis had united its marvellous energy to anæsthesia that the American practice of surgery underwent a complete revolution. We have, therefore, reserved a sketch of the history of this greatest of all American discoveries to the close of the *formative* and the beginning of the *practical* period, when through the combined influence of these agencies the practice of surgery was placed securely on a scientific basis.

The introduction of anæsthesia into the practice of surgery was not only the most notable achievement of American surgeons at that time, 1846, but, in its far-reaching influence upon the practice of surgery, anæsthesia has proved the most important evolutionary force hitherto discovered. The story of the struggle of the contestants for public recognition of priority in the discovery of anæsthesia forms one of the saddest and most revolting chapters in the history of the sciences. So fierce and relentless was the conflict that three of the four claimants became insane. Two of the latter were driven to suicide. Standing on the vantage-ground of half a century since the bitter contest closed, we are in a position to determine not only the part which each claimant had in the discovery of anæsthesia, but to whom the verdict of history awards the merit of introducing anæsthesia into the practice of surgery.

The term *anæsthesia* was suggested by Dr. Oliver Wendell Holmes, as appears from the following letter to Dr. Morton, dated November 21st, 1846: "Everybody wants to have a hand in the great discovery. All I will do is to give you a hint or two as to names, or the name to be applied to the state produced and to the agent. The state should, I think, be called anæsthesia. . . . The adjective will be anæsthetic. Thus we might say the state of 'anæsthesia,' or the 'anæsthetic state.'"

On the 30th day of March, 1842, Dr. Crawford W. Long, of Jefferson, Jackson County, Ga., removed a small glandular tumor from the neck of a patient, who had been rendered completely insensible by the inhalation of sulphuric

ether. The operation was completely successful, as the patient was not conscious of the procedure and made a good recovery. This was the first case of the employment of an anæsthetic in the practice of surgery recorded in modern surgical literature. It was not an accidental occurrence, but the result of careful observation and experiment in a truly scientific spirit. Dr. Long had witnessed the effects of nitrous oxide—laughing gas—in rendering persons insensible to painful injuries when under its influence, and, to satisfy himself of this fact, he took the gas himself, and received injuries that he was not conscious of until he recovered from the effects of the gas. These experiences induced him to undertake his first surgical operation while the patient was under the influence of the anæsthetic. Dr. Long continued to employ anæsthetics in his surgical practice for seven years, or until 1849, before he published an account of his discovery.

Fig. 27.—Crawford W. Long (1816–1878).

Crawford W. Long (1816–78) was born on the 3d day of November, 1816, in Danielsville, Madison County, Ga. He graduated from the University of Georgia in 1835, and from the Medical Department of the University of Pennsylvania in 1839. He began the practice of his profession at Jefferson, Jackson County, Ga. The operations which he performed were of a minor character, as there were no hospitals at that time accessible to him. He was also deprived of the advantages of medical societies and medical journals, but his success as a surgeon gave him a local reputation of the highest character. He died on the 16th day of June, 1878.

On the 11th day of December, 1844, Dr. Horace Wells, a dentist of Hartford, Conn., having observed that persons who took laughing gas and received injuries were unconscious of pain until they recovered from the effects of the gas, had one of his own teeth extracted while he was fully under the effects of the gas, and experienced no more pain than "the prick of a pin." On recovering, he exclaimed: "A new era in tooth pulling! It is the greatest discovery ever made." He introduced it into his dental practice and daily extracted teeth without pain. Impressed with the value of his discovery, in 1845 Dr. Wells visited Boston for the purpose of giving it greater publicity, but failed in awakening an interest in those he consulted. It is alleged that he attempted the use of sulphuric ether, but did not succeed in accomplishing any practical results with it.

On the 30th day of September, 1846, Dr. W. T. G. Morton, a dentist of Boston, Mass., administered sulphuric ether to a patient and extracted a tooth without pain. Morton had been a pupil, and subsequently a partner, of Wells,

and through the medium of these close relations the former had become familiar with the experiments and practice of the latter in the use of nitrous-oxide gas in dental operations. In attempting to repeat Wells's methods of practice, Morton found difficulty in securing a supply of gas, and applied to Dr. C. T. Jackson, a chemist of Boston and his former instructor. On learning what use Morton was to make of the gas, Jackson suggested the use of sulphuric ether, which would have the same effect, required no apparatus, was entirely safe, and was readily obtained. It was on this advice that Morton performed the operation of September 30th. In the belief that he had made a discovery of great pecuniary value, Morton took out patents, both in this country and in Great Britain, under the name "Letheon."

On the 16th day of October, 1846, Dr. John C. Warren, one of the surgeons of the Massachusetts General Hospital, removed a small vascular tumor from the neck of a patient, under the full influence of the "letheon," the identity of which Morton concealed by adding to the ether aromatic oils. The operation was performed at Morton's request, in order to test the value of the anæsthetic in surgical operations. Though the trial proved entirely successful, the effort of Morton to conceal the true nature of "letheon" prejudiced the surgeons against its further use until he acknowledged that the active agent in the preparation was sulphuric ether. The anæsthetic was then freely tested in capital operations, Dr. Warren resecting a lower jaw and Dr. Hayward amputating above the knee joint. The success of these operations, while the patients were under the influence of the anæsthetic, was so complete and satisfactory as to gain the applause of not only the eminent operators and surgical staff of the hospital, but of the entire medical fraternity of Boston. The Massachusetts General Hospital at once became a luminous centre, ushering in the dawn of the new era in the practice of surgery. Scarcely a half year passed before its rays illuminated every hospital in the capitals of this country and Europe, and anæsthesia in the practice of surgery was universally acknowledged as the greatest and most beneficent discovery in the annals of science.

Morton subsequently petitioned Congress for an allowance from the public treasury as the discoverer of anæsthesia in surgical operations, and thus brought under public discussion the question of priority. The friends of Long, Wells, and Jackson appeared before the committee of the House, to whom the matter was referred, and contested Morton's claims. Congress failed to take action, and the contest passed unsettled into history.

In the light of the preceding facts we conclude that Dr. Crawford W. Long, a surgeon, first used ether as an anæsthetic in the practice of surgery, but did not publish the fact until others had independently repeated his experiment. 2. Dr. Horace Wells, a dentist, was the second person to use an anæsthetic, but limited it to nitrous-oxide gas in dental operations. 3. Dr. W. T. G. Morton, a dentist, experimented to find an anæsthetic in dental operations, and was led to

use sulphuric ether at the suggestion of Dr. C. T. Jackson, a chemist. Morton succeeded so well that he concluded that he had made a discovery of great pecuniary value, and obtained patents. In order to give it publicity and repute in the profession, he solicited Dr. John C. Warren to use it in the Massachusetts General Hospital during an operation. 4. Dr. C. T. Jackson, a chemist, merely suggested that sulphuric ether is more readily used as an anæsthetic than nitrous oxide.

There is a monument standing in the Public Garden of Boston on which is inscribed the verdict of history as to the honor and glory of introducing anæsthesia into the practice of surgery:

> *To commemorate the discovery that the inhaling of ether causes insensibility to pain, first proven to the world at the Massachusetts General Hospital in Boston, October, A.D. MDCCCXLVI.*

Antisepsis in the practice of surgery had its origin with British surgeons. The principles on which its employment is based were scientifically established by Mr. Lister during the years 1870–75, and by him reduced to a definite system of practice which has been universally accepted. But, like all innovations upon long-established customs which are revolutionary in their operations, antisepsis was received by the older and more conservative surgeons of Europe with doubt and hesitation. But the American surgeons who visited Edinburgh and witnessed the practical application of antiseptics under the directions of Mr. Lister, and the remarkable healing of wounds without suppuration, were profoundly impressed with the far-reaching influence of the discovery upon the future practice of surgery, and hastened to introduce the new method into hospitals at home. The announcement of the results of antisepsis in the treatment of wounds was received in this country with genuine American enthusiasm, and the prescribed antiseptic agents were immediately placed on trial in scores of hospitals. The results justified the claims of visitors to the wards of the Edinburgh surgeon, and antisepsis took its rightful position as an indispensable factor in the American practice of surgery. And in the application of antisepsis to practice, the surgeons of no country have excelled the American in their efforts to adapt means to an end in the construction of operating-rooms and their equipment with every conceivable device to secure perfect asepsis of the patient, the surgeon and his assistants, the instruments, the wound, and its dressings. In hundreds of hospitals in this country the antiseptic treatment is carried out with such precision of details as to eliminate pus in operated cases. The results are simply marvellous. Operations that half a century ago were unthought of and even unthinkable on account of their danger, are daily performed with the most absolute success. In many operations which once had a high death rate the

mortality has been reduced so as to be merely nominal, and in a few once capital operations the death rate has been eliminated from the record altogether.

In the year 1826 Professor Sewall, of Columbia College, Washington, D. C., reviewing the progress of medicine in this country during the sixty years of its then national existence, spoke in the following eulogistic terms: "If in sixty years, with the limited means we have possessed and with all of the difficulties we have had to encounter, we have produced the best system of medical education, the most perfect system of medical police that has been exhibited to the world; if we have produced some of the best practical and elementary books, and some of the most eminent physicians and surgeons of any age or country; . . . what will be our advance in the sixty years to come?"

Those sixty years have passed, and an additional score of years have been added to the number, and how insignificant and even contemptible appear the system of medical education, the medical police, and the medical literature of that period! Then there were twenty medical colleges, giving instruction annually to about two thousand students; now there are one hundred and fifty-seven medical schools, educating upward of twenty-eight thousand students. Then the medical police was limited to gratuitous advice to the civil authorities; now it is a controlling force in the protection and promotion of the public health. Then but three surgical works had been published and but two medical periodicals were regularly issued; now forty-five native surgical works were published in two years and three hundred journals are regularly issued. Then there were three fully equipped hospitals; now they are found by the score in the large cities, and scarcely a village community is without its local hospital.

In concluding this sketch of the evolution of the American practice of surgery we have not sought to magnify the achievements of individual surgeons, nor even to enumerate what must be regarded as notable events in the general history of surgery, except so far as such achievements and events illustrate the elemental conditions and forces which governed its progress and development. For this reason we have dwelt more upon the special features, educational and experimental, of the early periods of our history than upon the triumphs of these modern times, which are but the fruitage of the culture of the past. What the American practice of surgery is to-day will be amply illustrated in these pages by surgeons whose daily duties are in the special fields of which they are the historians. The records of that practice will justify the conclusion of Mr. Erichsen in the paper referred to: "I know no country in which, so far as it is possible to judge from contemporary medical literature, there is so widely diffused a high standard of operative skill as in this country."

PART I.

SURGICAL PATHOLOGY.

INFLAMMATION.

By ALDRED SCOTT WARTHIN, Ph.D., M.D., Ann Arbor, Michigan.

I. GENERAL CONSIDERATIONS.

1. Inflammation is a Pathological Complex, Essentially Adaptive, Protective, and Reparative, Constituting the Reaction of the Body Cells to Injury, either Direct or Referred.

Experimental and comparative pathology have given us a broad biologic conception of the reaction of cells to injury. From the lowest forms of animal life up through the higher to man we find that tissue injury, when not so severe or extensive as to cause the death of the individual, excites a definite response in the animal organism. No matter what the nature of the harmful agent ("irritant") may be, this reaction on the part of the damaged organism, unicellular or multicellular, is in its essence the same; that is, it is at foundation an attempt to oppose or evade the irritating agent, to counteract its harmful effects, and to repair the damage caused by it. Naturally, these protective and reparative efforts are carried out somewhat differently in the case of different animals, according as their structure is simple or complex. Likewise the great variety of injurious agents and the varying conditions under which they act must influence the course of the reaction. It may be stated also, in the beginning, that these protective and reparative processes are often inadequate or imperfect; in fact, in the attempt to protect itself the organism may inflict further damage upon itself, even to such an extent that the death of the individual may ensue. In spite of these imperfections the essential fact remains—the process of inflammation is at bottom protective and reparative.

In the case of unicellular animals the protective process can be studied in its simplest form. In such an animal both protective and reparative functions are reduced to the basis of the single cell. It may protect itself by the extrusion or destruction of the harmful agent, the latter event being brought about by means of intracellular chemical processes akin to digestion; while cell defects due to the action of the irritant are repaired through a new growth of cell substance.

In multicellular animals the division of labor among different cell groups results in the assignment of protective and reparative functions especially to certain kinds of cells, and the more complex structure of the organism necessitates a much more elaborate method of protection. While individual cells

retain to a greater or less degree the individual functions of the unicellular organism, these become lessened or may be wholly lost as the cell gains in specialization. Accordingly, in the higher vertebrates and in man we find that the removal and destruction of harmful agents are effected chiefly by wandering mesoblastic cells, lymphoid tissue, and the endothelium; while repair is chiefly brought about by the proliferation of fixed connective-tissue cells and endothelium. The more highly developed structure of the multicellular organism, its complex nutritive mechanism, and the important part played in the body economy by the vascular system cause the involvement of the latter to assume a very important rôle in the processes both of protection and of repair. Further, the influence of the nervous system is also a factor of great importance in connection with these processes.

If we should select the most constant and characteristic phenomenon of the reaction to injury in multicellular organisms it would be found to lie in the assemblage of cells of the leucocyte type at the site of injury. These cells, indeed, may be regarded as analogous to unicellular organisms, and they present the protective functions characteristic of the latter. The difference in reaction to injury between unicellular and multicellular organisms is the result simply of the specialization of function and the more complex mechanism of the latter. It becomes clearly evident, therefore, that the essential principles underlying the response of the animal organism to injury are the same for all forms of animal life from the lowest to the highest. And it is to this reaction of the animal organism to injurious agents and the lesions produced by them that we now, in accordance with the majority of modern pathologists, apply the term *inflammation.*

2. Differences of Conception of the Inflammatory Process.

The earliest conception of inflammation (*inflammatio, phlogosis*) was a purely clinical one. At the beginning of the Christian era the term was applied by Celsus to local changes in the superficial portions of the body characterized by redness (*rubor*), swelling (*tumor*), heat (*calor*), and pain (*dolor*). Since these phenomena appeared constantly as the results of certain injuries and irritants to the external portions of the body Celsus designated them as the four cardinal symptoms of inflammation. Later, a fifth symptom of disturbed function (*functio læsa*) was added. While this primitive definition still exercises a traditionary influence, the term inflammation has gradually come to include a large number of pathological conditions of the internal organs believed to be analogous to the inflammatory process, as well as all those morbid processes which in etiology and course cannot be separated from, and which pass insensibly into, conditions showing the classical symptoms. The term, therefore, gradually came to be applied to conditions in which some or all of the cardinal signs were

absent or could not be recognized, and the purely clinical significance at first attached to it was weakened or lost. Such a usage may be taken as an indication of the awakening realization that all the processes included under the term were in essence of the same nature and significance. And that this latter is a fact has been demonstrated beyond all doubt by the study of the minute changes in the organs and tissues in and about the inflamed area and by the results obtained through experimental and comparative pathology.

From Boerhave, who regarded inflammation as the result of stasis, to Rokitansky, who emphasized the vascular dilatation, slowing of the blood stream, and serous exudation, to Virchow, who regarded inflammation as an overstimulation of the functional, nutritive, and formative irritability of the cells, and finally to Cohnheim, who first studied the phenomena of inflammation in the living animal directly under the microscope and was thus enabled conclusively to demonstrate that the cardinal changes are vascular disturbances leading to emigration of the white cells, serous exudation, and diapedesis of red cells, there may be traced a constant widening of the field covered by the designation inflammation, so that the term came to be applied to the great majority of pathological processes in the body without reference to the original symptomatic significance. But even Cohnheim was unable to see anything in the inflammatory process of service to the body; its chief significance to him lay in the primary lesion of the blood-vessels permitting the passage of the blood elements. While giving to pathology the important knowledge of the vascular alterations occurring in the inflammatory process, Cohnheim threw no light upon its essential nature. His views, however, were for a long time accepted as the most satisfactory interpretation of the inflammatory phenomena, and they still influence greatly some of the leading pathologists of the present day.

During the last several decades efforts have been constantly made to ascertain the common feature of the various processes classed as inflammatory, with the view of arriving at a fundamental conception of the inflammatory process. The varied etiology and the very different clinical and histological pictures presented by different inflammations afforded no basis for a fundamental definition. In the effort to make of inflammation both a clinical and a pathological entity the term itself fell somewhat into disrepute with both clinicians and pathologists, and it was even proposed by some (Thoma, Andral, and others) to drop it altogether.

But a new conception of the process was slowly evolving—one which would be able to harmonize all facts, remove all difficulties, and give to inflammation an entity, not as a condition or state, but as a process having in all of its manifold manifestations one essential unity, viz., that of protective and reparative reaction to injury. This new conception may be said to have had its origin with the discovery of karyokinetic cell-division and the demonstration that in practically all inflammatory lesions cell proliferation occurs to a greater or less de-

gree. At first, this new formation of cells was regarded as a sequela of inflammation rather than as an essential part of the process. But it was soon discovered that other features of the inflammatory process could likewise be demonstrated to be protective in character.

To Metschnikoff do we owe the knowledge that the assemblage of wandering cells at the point of injury may precede and be independent of the vascular changes, and that this phenomenon constitutes the most characteristic and constant factor of the reaction to injury. Regarding this collection of wandering cells as primarily intended for the exercise of their function as phagocytes, Metschnikoff formulated a new conception of inflammation as a reaction of phagocytes against the injurious agent. The essential and fundamental element of inflammation is, then, a means of defence for the animal organism. While the new conception of inflammation as adaptive, protective, and reparative owes more to Metschnikoff than to any other modern investigator, yet his efforts to establish phagocytosis as the essential element of the process must be regarded as based upon a too narrow conception. That phagocytosis is only one of the protective functions of the body-cells exercised in inflammation was clearly recognized by other investigators, and the lines of work followed in opposition to Metschnikoff's views have served to give a still firmer and broader foundation to the new conception.

The demonstration of the active participation of the blood-vessels and endothelium (Klebs, Heidenhain, and others), the influence of the nervous system (Samuel), the part played by chemotaxis (Bordet, Leber, Buchner, Gabritschewsky, and others), and the presence in the blood and serum of antibacterial bodies (alexins) (Nuttall, Buchner, and others) served further to establish the new conception more securely. It has been shown also that leucocytes contain or produce bactericidal substances (Buchner, Hankin, Bordet, Stokes and Wegefarth, Loewit, and others). The bactericidal action of the serum in inflammatory processes has been conclusively demonstrated, as has also the mechanical protection afforded by the cellular infiltration, fibrinous exudate, granulation-tissue, etc. The function of the fluid exudate in diluting or washing away injurious agents may also be mentioned as one of the protective factors of minor importance. Lastly, but constituting one of the most important factors in the body's defensive processes, is the production by the body-cells of antitoxins whereby injurious chemical agents are neutralized or destroyed. Ehrlich's theory serves to illuminate the processes of local inflammation as well as of general infections and intoxications.

Summing up, then, all the factors of the inflammatory process and viewing them in the light of modern research we see that in all animals and in response to all kinds of injury they are essentially the same, though often varying in proportion, and that their unity lies in a constant tendency toward protection and repair. Inflammation, therefore, as defined above, can be regarded only as a

process-complex essentially adaptive, protective, and reparative, called into action by a primary tissue lesion.

To this view nearly all the pathologists of the present day accede. A few examples of recent definitions may be given here:

Inflammation is the series of changes constituting the local manifestation of the attempt at repair of actual or referred injury to a part, or, briefly, as the local attempt at repair of actual or referred injury.—ADAMI.

Inflammation is a local reaction, often beneficial, of the living tissue against the irritating substance. This reaction is produced chiefly by phagocytic activity of the mesodermal cells. In this reaction there may, however, participate not only changes of the vascular system, but also the chemic action of the blood plasma and tissue fluids in liquefying and dissolving the irritant agent.—PODWYSSOZKI.

Inflammation is the reaction of the tissues to local injuries calling forth protective measures; an imperfect pathologic adaptation, often leading to consequences that are dangerous *per se* and may defeat its purpose.—HEKTOEN.

On the whole the processes involved in inflammation are conservative, and, within the limitations which may be set by the varied and changing conditions of injury, tend to maintain the welfare and sustain the life of the individual.—DELAFIELD *and* PRUDDEN.

The reaction of the organism against injurious agents. . . . Local inflammation may be regarded as an increased tissue function which is also active under normal conditions but of so slight a degree as not to be perceived.—RIBBERT.

Only a few pathologists, among these Ziegler, still emphasize the tissue lesion, particularly the vascular alteration, as the most essential feature of the inflammatory process. But among surgical writers we find a small number, who, following Hueter's dicta, assert that the term inflammation should be applied only to the processes caused by pyogenic micro-organisms; that is, to suppurative processes. This confusion of inflammation and pyogenesis is illogical and unfortunate. There is no etiological entity to which the application of the term inflammation can be restricted. Physical, chemical, and thermal agents can produce precisely the same changes as those seen in local infections. On the other hand, infection may occur without inflammation. The fact that purulent inflammations are the most common and important forms of the process falling within the province of practical surgery gives no warrant for the usurpation by this branch of medicine of the term inflammation for one particular manifestation of the inflammatory process. The term has been too long applied to various other processes of essentially the same nature and significance to permit of such a narrowing of its meaning. If the phenomena of the local reaction of the body to injurious agents are in essence the same, no matter what the etiological agent may be, the term inflammation must include them all. That they are the same, in all essential respects, will be shown in the following paragraphs.

3. The Reaction of the Tissues to Injury.

Injury to Non-vascular Tissues.—The cornea offers itself as suitable material for the study of the effects of injury upon non-vascular tissues. According to Senftleben and others, if the centre of the rabbit's cornea be touched with a strong solution of zinc choloride without causing an actual break in its continuity, there is a necrosis of the corneal cells at the point of application, with the development of an encircling zone in which the corneal cells are enlarged, granular, and tumefied. There is apparently no increase of wandering cells and the necrosed cells are replaced through the multiplication of the neighboring hypertrophic cells. The two essential features here are cell necrosis and cell division; and such a process must be regarded as the simplest form of an inflammation. It is doubtful, however, if such a simple form actually occurs under such conditions. It is almost impossible to repeat Senftleben's results; in the great majority of cases, if not in all, the slightest perceptible injury to the cornea causes an increase in the number of wandering cells in or about the damaged area. As the neighboring vessels of the conjunctiva may show no perceptible changes, it is most probable that the wandering cells come to the damaged area out of the lymph spaces of the surrounding tissue (see Fig. 28).

If a simple cut be made into the cornea, either with or without previous cauterization, practically the same changes are seen. There occurs within a short time a collection of leucocytes in and about the point of incision. At first, these are apparently attracted from the surrounding tissue, as the blood-vessels of the conjunctiva may show no changes. Cell division takes place and repair is effected. The inflammatory process consists, then, essentially of cell injury, positive chemotaxis, and cell proliferation. When the degree of the trauma is greater (repeated trauma or cauterization, repeated application of toxin), the neighboring vessels of the conjunctiva become involved, as shown by their dilatation, increased number of leucocytes, and leucocyte emigration, as well as by serous exudation. The leucocytes mass themselves in greater numbers about and in the injured area. The number may become so great that the tissue presents the appearance of a purulent infiltration. Liquefaction necrosis may take place (repeated application of chemicals or toxins), and the histological picture produced may be exactly similar to that following the growth of pyogenic micro-organisms.

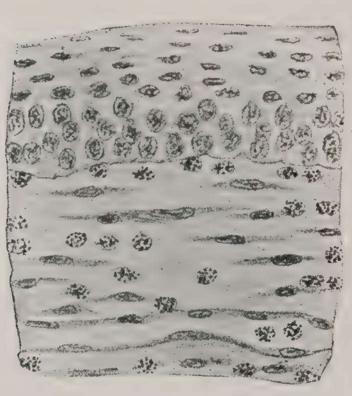

Fig. 28.—Inflamed Cornea. Hypertrophy of corneal cells; assemblage of leucocytes. (*After Ribbert.*)

If pyogenic bacteria are injected into the centre of the cornea and multiplication of the organisms follows, there is first seen around the growing colony

a zone of degeneration and necrosis of the corneal cells. Within a short time leucocytes collect about the damaged area, coming first from the corneal tissues and later from the blood-vessels at the periphery of the cornea. These vessels are found to be dilated and containing an increased number of leucocytes. They present also evidences of serous exudation and emigration of the leucocytes. That the leucocytes come chiefly from the vessels is shown at a certain stage by their greater number at the periphery of the cornea and their gradual approach toward the area of injury. Finally, large numbers of leucocytes may be collected in the lymph spaces about the tissue lesion. If the bacteria introduced were of a low virulence, or if the body's resistance is sufficient to inhibit the growth of the colony or to kill it, proliferation begins and repair is effected. The inflammatory process under such circumstances in no way differs from that produced by other agents.

If the micro-organisms injected are virulent and continue to develop, the

Fig. 28, A.—Corneal Suppuration. *a*, Limbus corneæ with hyperæmia of ciliary vessels and purulent infiltration; *b*, remains of epithelium; *c*, purulent infiltration of cornea; *d*, necrosis; *e*, pus; *f*, Descemet's membrane; *g*, endothelium of anterior chamber. (*After Weichselbaum.*)

area of necrosis and degeneration widens, more leucocytes come into the damaged area, the lymph spaces contain more fluid, and the peripheral vessels show a more marked reaction. Many of the leucocytes are seen to be acting as phagocytes, having taken up large numbers of the invading organisms. After a time the leucocytes massed in the central portion of the necrotic area undergo degeneration, and a liquefaction of the infiltrated necrotic centre follows, leading to the formation of an ulcer (see Fig. 28, A). The virulence of the infective

agent may now be weakened, or the latter may be restrained from further growth by the leucocyte barrier. Proliferation of the neighboring corneal cells may take place, the dead tissue and leucocytes being either cast off or organized, and the ulcer finally becomes completely healed. In the process of repair new blood-vessels may grow into the cornea from the blood-vessels of the surrounding tissues, and the previously avascular tissue then becomes vascularized. Should the organism be unable to overcome the virulence of the infective agent, the area of necrosis and degeneration increases and the inflammatory process extends.

Injury to Vascular Tissues.—The skin or mesentery of experimental animals may be utilized for the demonstration of the changes set up by injury to vascular tissues. If the slightest possible injury that can be recognized be produced in such tissues by means of heat, chemical action, or aseptic incision, practically identical changes, varying only in degree, will be found at the point of injury. The simple aseptic incision may be taken as an example. The solution of continuity of the incised part leads at once to a necrosis or degeneration of the cells along the line of incision. The edges of the wound are almost immediately glued together by serum or blood clot, and there is a slight serous infiltration of the tissue bordering upon the incision. The tissue cells in the immediate neighborhood of the cut enlarge and send out processes into the exudate, binding the sides of the wound together. Protoplasmic bridges may thus be formed across the wound. At the same time the neighboring capillaries show slight or moderate dilatation, and there is a slight increase of wandering cells. Later, cell proliferation with a new formation of capillaries takes place and the tissue continuity is again restored. In the epidermis the reaction to the injury is shown simply by a new formation of epithelial cells. There action to slight injury in vascular tissues is characterized, therefore, by slight vascular changes, emigration of leucocytes, and exudation, the chief factor of the reaction being progressive changes (hypertrophy and proliferation) of the tissue cells. There is, then, no essential difference in the reaction to slight injury in avascular and vascular tissues.

If the degree of injury be more marked, so that the tissue lesion (degeneration and necrosis) is more extensive, there is a more pronounced and rapid reaction on the part of the blood-vessels. These show a marked dilatation, with slowing of the blood stream, marginal disposition of the leucocytes, increased formation of lymph, and emigration of leucocytes from the vessels. Red blood cells may also escape. The leucocytes collect in the tissue spaces in or about the injured area (see Fig. 29). When the process is on the superficial portions of the body the classical symptoms may be present. According to the nature of the irritant, its virulence, and the resisting powers of the organism, the inflammatory process may continue to extend or cell proliferation may begin, the exudate disappears, the vessels resume their normal condition, and all traces of the injury and the reaction pass away.

Should the injurious agent be one capable of unlimited growth and continuous injury to the body tissues, as is the case with virulent pathogenic organisms, the pus cocci in particular, the tissue lesion is more severe and assumes a progressive character, while the inflammatory reaction is proportionately more marked. When the focus of irritation is situated in the superficial parts of the body the classical symptoms are usually clearly defined. On microscopical examination precisely the same essential changes are found as in the non-infective inflammations, the only difference being one of intensity and adaptation to the different character of the injurious agent. At the point of a pyogenic infection there is produced by the growing colony of bacteria a tissue degeneration or necrosis. About this lesion there is a rapid and marked vascular reaction. Within a few hours usually the vessels are found to be markedly congested, packed with red blood cells, and showing a marginal disposition of

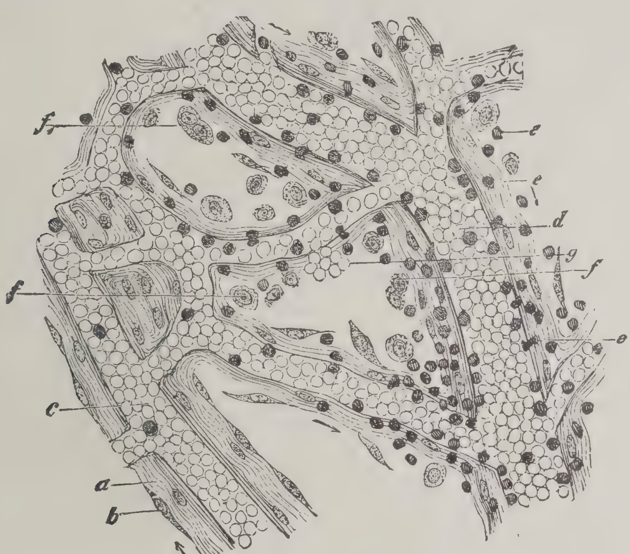

Fig. 29.—Inflamed Human Mesentery (osmic-acid preparation). *a*, Normal trabecula; *b*, normal epithelium (endothelium); *c*, small artery; *d*, vein with leucocytes arranged peripherally; *e*, white blood cells, which have emigrated or are emigrating; *f*, desquamating endothelium; f_1, multinuclear cells; *g*, extravasated red blood cells. × 180. (*After Ziegler.*)

the leucocytes, which are greatly increased in number. In the tissues the number of wandering cells is greatly increased. The mononuclear forms are very numerous and often predominate. Both leucocytes and tissue cells acting as phagocytes and containing numbers of the bacteria may be found. As a rule, the leucocytes in the immediate neighborhood of the growing colony are multi- or polymorphonuclear, while in the outer zone of the infiltration the mononuclear forms prevail.

As the process advances, the damaged area becomes densely infiltrated with leucocytes, the dead tissue elements undergo liquefaction, while the leucocytes in the area of softening show signs of degeneration. The process has now reached

the stage known as *suppuration,* and there is formed an *abscess* more or less sharply defined from the surrounding tissues (see Fig. 29, A). If on a surface, the suppuration leads to a superficial loss of substance—an *ulcer.* The mass of leucocytes occupying the central necrotic area constitutes *pus.* In this pus the great majority of the leucocytes are found to be phagocytes—that is, they contain numbers of the infective organism. About the border of the pus area the tissues are swollen, more or less infiltrated, the blood-vessels congested, and there is more or less hemorrhage. At this stage no evidences of cell proliferation can usually be found. If the infective agent has great virulence, the process may continue to extend indefinitely with a repetition of the same phenomena, and finally become generalized, or the organism may succumb. If the virulence is overcome and the colony of bacteria dies out or ceases to reproduce,

Fig. 29, A.—Small Abscess in Heart Muscle. Colony of pyogenic cocci in centre of necrotic area, which is surrounded by a zone of leucocyte infiltration. (*After Ribbert.*)

cell division begins about the border of the abscess, new capillaries extend into it, and the area is gradually replaced by new tissue.

The same picture of suppuration may be produced by the injection, into the tissues, of certain chemical irritants, such as turpentine, mercury, petroleum, creolin, bacterial toxins, etc. Inasmuch as under such circumstances there is no continuous production of irritating substances, as in the case of bacterial infection, the purulent reaction thus produced lasts for a shorter time, cell proliferation begins more quickly, and healing is attained in less time. Continuous injections, however, will cause a progressive purulent process, not to be distinguished in any way from that due to bacterial infections, in so far as the local phenomena are concerned.

In the case of certain injurious agents the inflammatory reaction is characterized by serous exudation rather than by leucocytic. Others still are characterized by the production of a fibrinous exudate. The same agent may at one time produce a reaction characterized by suppuration, at another time one characterized by serous exudation, and under other conditions it may give rise to an exudate consisting chiefly of fibrin. These variations in the inflammatory reaction are especially characteristic of those inflammations which are due to infections. The factors modifying the nature of the reaction are many: the nature of the injurious agent, the location and character of the injury, the

degree of intensity, the general and local conditions of the organism, etc., all of which serve to give to the inflammatory reaction a varied clinical and pathological picture.

Nevertheless, as we have seen above, there is a unity in the reaction to injury which makes of the inflammatory process a distinct entity, no matter what the nature of the injurious agent or the conditions under which it acts. Whether traumatic, thermal, chemical, or infective in etiology, or formative, serous, purulent, or fibrinous in character, the process has the entity of adaptation, protection, and repair against injury. As Ribbert and others have pointed out, inflammation may be conceived of as a body function—the function of protection, and comparable to the other body functions. Cell proliferation, chemotaxis, emigration of leucocytes, phagocytosis, the production of antibodies, etc., are probably always taking place in the body to some extent, but unnoticed. Only when the demand made upon this function is so great as to become locally prominent does it become manifested in the form of an inflammation. If it be urged against this view that the inflammatory process often in itself is a source of danger to the organism, the same might be applied to other functions—for instance, the digestive, the products of digestion often becoming factors in auto-intoxications. Viewed broadly, inflammation is to be regarded as a phylogenetic evolution, developing as have all the other functions of the organism.

4. Etiology of Inflammation.

The inflammatory process has no etiological entity. The causes of inflammation are not specific. Any injurious agent may produce inflammation, provided its action is not so severe as to kill the organism or the tissues *en masse* or to inhibit the function of protection and repair. Extrinsic agents—such as mechanical, thermal, chemical, electrical, radio-active, infective, etc.—are among the most common causes of inflammation. In surgical practice it is chiefly with these extrinsic causes that the surgeon has to deal. But injurious agents capable of exciting inflammation may be produced within the body as the result of disturbed metabolism, disordered function, etc. The anæmic necrosis of tissue areas due to local obstruction of the blood-vessels excites also an inflammatory reaction in the neighboring living tissues.

In the great majority of cases the inflammatory reaction is probably toxic in origin. The irritant poison is chiefly, as far as surgery is concerned, bacterial. In general medical work inflammatory processes due to auto-intoxications occupy also a prominent position.

The agents causing inflammation may act upon the body from without (*ectogenous inflammation*) or through the lymph (*lymphogenous*) or the blood (*hæmatogenous*). When the avenue of entrance of the injurious agent (bacteria) is not known, the inflammation is styled *cryptogenic*.

The inflammatory process may spread by direct extension (*inflammation by continuity*), or the injurious agent may be transported through the lymph or blood stream to other parts of the body, there to excite new inflammatory foci (*metastatic inflammation*). Through the excretion of poisonous substances the excretory organs may become the seat of inflammatory processes (*excretory inflammation*).

The action of the harmful agent may be very transitory or it may be prolonged through some period of time. Particularly is this the case with infective agents. Through the continuous new formation of poisonous substances by the colony of living parasites, an inflammatory reaction may be kept up almost indefinitely until the organism finally conquers or succumbs. The action of the inflammatory agent may be so slight as to produce a lesion that is clinically unrecognizable, and even on microscopical examination the evidences of damage to the tissue are with difficulty made out. At other times the action is so intense that extensive lesions easily seen by the unaided eye are produced.

Usually the inflammatory reaction quickly follows the injury, the interval of time varying from a few hours to a few days; but in some instances the reaction is long delayed. The character of the exciting cause, its virulence, the conditions of nutrition about the damaged area, the influence of the nervous system, etc., are probably the chief factors causing a delayed reaction. Some agents may inhibit the protective function or even cause destruction of some of the elements therein concerned. Roentgen rays, for example, will cause a disintegration of the leucocytes in the irradiated area and also inhibit cell proliferation. These facts may explain in part the long-delayed inflammatory reaction seen after repeated exposure to the rays.

5. The Factors Concerned in the Inflammatory Process.

In the surveyal of the course of inflammatory processes in general, the various associated phenomena are found to fall into several more or less well-defined

The Factors Concerned in the Inflammatory Process.

1. Effects of Injurious Agent. Tissue-Lesion.	2. Reaction. Protective and Defensive.			3. Resolution. Reparative.
a. Disturbance of cell relation. *b.* Solution of continuity. *c.* Tissue degenerations. *d.* Necrosis.	Chemotaxis. Leucocytosis. Emigration of white cells.	Vascular disturbances. *a.* Congestion. *b.* Stasis. *c.* Leucocytosis. *d.* Marginal disposition. *e.* Emigration. *f.* Serous exudation. *g.* Diapedesis. *h.* Thrombosis.	Phagocytosis. Chemical protection. *a.* Antibacterial. *b.* Antitoxic. Mechanical. Diluent. Irrigant.	*a.* Cell-proliferation. *b.* Regeneration. *c.* Organization. *d.* Cicatrization.

groups, the first of these being the immediate results of the harmful agent, the second consisting of factors essentially protective and defensive, while the third group is made up of the formative and reparative factors. The general process of the reaction to injury having been sketched above, it may now be profitable to consider more in detail the most important factors of these groups.

Tissue-Lesion.—Although the primary tissue-lesion is the cause and not a part of the inflammatory reaction, it is difficult practically to separate it from the phenomena which are immediately dependent upon it, and it is, therefore, usually included in a general survey of the process of inflammation. Moreover, from the clinical side the changes occurring in the tissues, either primarily as the direct result of the action of the etiological agent or as secondary to the inflammatory process itself, are of the greatest practical importance. The primary tissue lesion varies with the nature of the etiological agent, the intensity of its action, the location of the injury, the condition of the tissues, the general state of the organism, etc. At times it is so slight as not to be recognizable either with the naked eye or microscopically; at other times so severe or extensive as to be recognized easily macroscopically, and clinically to attract the chief attention. This is particularly the case when the injury affects a large portion of the tissues, as in the case of extensive burns, corrosions, freezing, or severe and widespread infections. In the case of bacterial infections the tissue lesion may assume a progressive character, involving large areas by direct extension.

The primary tissue lesion may be simply a disturbance of cell relationship or a solution of continuity, or it may show itself in the form of any one of the varieties of tissue degenerations or necrosis. The acute parenchymatous degenerations, cloudy swelling, hydropic degeneration, fatty degeneration, mucoid degeneration, etc., and the various forms of necrosis, simple, coagulation, liquefaction, and gangrenous, are the most common tissue lesions exciting the inflammatory reaction. While in some inflammations the tissue lesion may be overshadowed by the phenomena of the reaction, in others it occupies the most prominent position both clinically and microscopically.

In the great majority of cases the tissue lesion involves the blood-vessels in or about the site of injury, but, as we have seen, such vascular changes are not necessarily a part of the inflammatory reaction—as, for example, in the corneal change following slight injury. Inasmuch as the vascular changes may take place at a distance from the actual seat of injury, the participation of the vessels in the reaction must be explained, in such cases at least, upon other grounds than that of a direct injury to the vascular walls. There is good reason for believing that the walls of the blood-vessels play chiefly an active part in the inflammatory process, and not merely a passive one due to injury.

Secondary injury to the tissues is often added to the primary lesion as the result of the disturbances of circulation and nutrition and the collection of

exudates either in the tissue spaces or in the body cavities. To this secondary damage the inflammatory process often owes its clinical importance. Moreover, the dead and dying tissue acts as a further source of injury to the surrounding healthy tissue, in that it may give rise to chemical products which are irritant, and so extend the zone of damage. This is most likely to occur in the early stages of necrosis; later, the dead tissue becomes chemically indifferent and acts simply as a foreign body.

The Protective Factors. *Chemotaxis.*—As shown above, the chief phenomena of the reaction in the simplest forms of inflammation are the collection of wandering cells at the site of injury and the proliferation of the tissue cells. This assemblage of amœboid cells about the tissue lesion and the injurious agent precedes the vascular changes, if the process be viewed from the standpoint of its evolution. The attractive force exerted upon the amœboid cells is known as *positive chemotaxis.* It lies in diffusible substances produced in the injured area, through altered nutrition, the death of the tissue cells, or by bacteria. Such substances attract the amœboid cells and cause their assemblage at the site of injury. Negative chemotaxis may also occur, but positive chemotaxis, although varying in degree, is practically the rule in the inflammatory reaction. Particularly in the case of infective inflammations is positive chemotaxis marked, the great majority of pathogenic micro-organisms producing substances causing the amœboid cells of the body to move toward the bacteria. Chemotactic influences may also be exerted upon the white cells while still in the blood-vessels, and in part at least may account for their occurrence in greater numbers in the vessels of the damaged area as well as for their emigration. Such influences also direct the movements of the white cells after they have passed out of the vessels. Ordinarily the leucocytes leaving the vessels wander in the direction of the lymph stream. In the case of tissue injury they wander toward the site of the lesion and the injurious agent. About the latter they collect in masses and remain in its immediate vicinity.

Chemotaxis brings the amœboid cells to the point where their phagocytic function may be used to some avail. Not only upon the leucocytes and lymphocytes are such influences exerted, but also upon the endothelial cells of the vessel wall and upon the formative cells arising through the proliferation of the fixed connective-tissue cells. Whether regarded as a purely accidental phenomenon or as an attribute of the injurious agent and in itself ultimately injurious, the fact remains that it becomes a force aiding in the defence and protection of the body.

Vascular Changes.—As the vast majority of inflammatory processes occur in vascular tissues, the involvement of the blood-vessels plays a very prominent rôle, both clinically and pathologically. The first noticeable feature of the vascular involvement is an active hyperæmia, the arteries being dilated and the rate of the blood current increased. In a very short time, however, there is a

marked slowing of the blood stream, while the hyperæmia remains or increases, the capillaries and veins becoming greatly dilated. The rate of the blood flow may be still further diminished and very irregular. In portions of the damaged area a condition of stasis may exist. Thrombi may be formed in the veins. In the capillaries about the irritant (usually when infective) there are sometimes formed masses made up of agglutinated red blood cells (*agglutination thrombi*).

From the very beginning of the reaction the leucocytes increase in numbers in the vessels of the area, and as the blood stream becomes slowed their number is augmented until a condition of a more or less marked local leucocytosis exists. At the same time the viscosity of the white cells appears to be increased, since they collect in numbers along the vessel wall, where they remain adherent or move along but very slowly (*marginal disposition of the leucocytes*).

The leucocytes adherent to the vessel walls now begin to pass out in numbers (*emigration of the leucocytes*). This process is accomplished by the protrusion of a cell process (pseudopodium) through the intercellular substance between the endothelial cells. The cell protoplasm follows or "flows" after, until finally the entire cell has passed outside. Soon numbers of leucocytes collect outside the vessel and pass thence to the site of the injury or the injurious agent. Through the openings in the intercellular cement substance produced by the passage of the leucocytes the red blood cells may passively escape (*diapedesis*). Hemorrhage *per rhexin* may also occur as a part of the inflammatory process, the rupture of the vessel being due either to extreme dilatation or to its direct involvement in the tissue lesion.

At the same time there is an increased formation of lymph, the fluid collecting in the tissue spaces as an *inflammatory œdema* or passing out upon a free surface as an *inflammatory exudate.* From the normal lymph and blood plasma the inflammatory exudate differs in its albumin content, its frequently high fibrinogen content, as well as by differences in the amount of salts contained. Emigration and exudation do not always go hand-in-hand. Some inflammatory processes are characterized by a fluid exudation, others by a cellular. The passage of the fluid cannot, therefore, depend wholly upon the spaces left after the emigration of the leucocytes. Further, the difference in composition of the exudate and the blood plasma must also be taken as evidence of a different mode of origin. The chemical composition of the exudate also varies in different parts of the body.

The significance of the vascular changes just mentioned has been the object of a vast deal of research and discussion, but we are still ignorant of much concerning these processes. The modern view, however, tends steadily toward the belief that they are not simply the result of a passive lesion of the vessel walls due to the harmful agent, but that the vascular participation, particularly the part played by the capillary endothelium, is essentially an active process. The

increased permeability of the vessel wall may be due to an active contraction of the endothelial cells instead of a passive stretching and thinning. During inflammation the endothelial cells of the inflamed area become larger and more prominent; they also have the power of throwing out protoplasmic processes and of phagocytosis. Foreign bodies and bacteria may be taken up by them. They appear to be increased also in viscosity during inflammation, and the slowing of the blood stream and the marginal disposition of the white cells may be aided by this. Further, the character of the inflammatory exudate is strong proof that it is not simply a filtrate but a secretion produced by the selective activity of the endothelial cells. The influence of the vasomotor nerves upon the course of the inflammation may be taken as further proof of this view. Although the injurious agent may cause passive changes in the vessel wall, there are abundant reasons for believing that the vascular endothelium actively participates in and favors the occurrence of the inflammatory process.

The Cells of the Inflamed Area.—The cellular infiltration in and about the seat of injury is of a less simple nature than was formerly believed. More detailed studies of the cells composing it show that the problems concerning the origin, nature, and function of the round cells of the infiltration are not so easy of solution as was formerly believed. Consequently much discussion has been waged over these questions. While they cannot as yet all be answered positively, we have made at least some progress in our knowledge concerning the character of the cellular infiltration or exudate. In general, it may be said that the predominating forms of cells assembling at the damaged area vary according to the nature and location of the injury. As a rule, five types of round cells are characteristic of the inflammatory reaction—the finely granular oxyphile (neutrophile) polymorphonuclear or polynuclear, the coarsely granular oxyphile (eosinophile), the small lymphocyte, the large lymphocyte (hyaline cell), and the plasma cell. These different types may not always be present at the same time and in the same proportions. Although at the height of the reaction the inflamed area is usually rich in cells, the variety and proportion in a given case may vary greatly (see Fig. 30).

In experimental work it has been found that the first cells to move toward the tissue lesion are the eosinophile wandering cells. The neutrophile polymorphonuclear then respond, and at the same time the neutrophile polymorphonuclears in the blood-vessels begin to emigrate. As a rule, the cells of the infiltration during the earlier stages of inflammation are chiefly the neutrophile polymorphonuclears or polynuclears with an occasional eosinophile. Under certain conditions the number of eosinophiles may be very great or this form may even predominate.

As the inflammatory process increases numerous cells of the lymphocyte type appear, at first collecting about the periphery of the inflamed area, while the polymorphonuclear cells mass themselves in the centre about the inflam-

matory agent. Sometimes the lymphocyte type prevails from the beginning of the process, but in the majority of cases the number of lymphocytes increases as the course of the inflammation becomes more protracted. The source of the cells of the lymphocyte type has been a subject for much speculation. Their emigration from the blood-vessels has been denied by most writers, but according to recent studies such a process undoubtedly occurs. Nevertheless, it is most probable that the chief portion of the cells of the lymphocyte type found in the inflammatory infiltration does not come from the vessels, but arises in the tissues through cell division of pre-existing lymphoid cells found there. The writer agrees strongly with Ribbert as to the source of the lymphocytes.

As a rule, the lymphocytes usually appear in such numbers as to be easily recognizable only after several days from the beginning of the inflammation. They may be scattered among the polymorphonuclear leucocytes, or, as is more common, grouped in little collections. Ribbert regards these lymphocyte

FIG. 30.—Isolated Cells from a Granulating Wound (picrocarmine). *a*, Uninuclear leucocytes; a_1, multinuclear leucocyte; *b*, different shapes of uninuclear formative cells; *c*, double-nucleated formative cells; c_1, multinucleated formative cells; *d*, formative cells in the process of tissue-formation; *e*, completed connective tissue. × 500. (*After Ziegler.*)

groups as representing rudimentary lymph nodes, which under the influence of the inflammatory reaction become hyperplastic either from increased cell proliferation or as the result of an increased number of lymphocytes coming to them from the blood-vessels. That such rudimentary lymph nodes exist everywhere throughout the body there can be no doubt. Recent studies have shown their existence in practically all organs and tissues. In chronic inflammation the increasing hyperplasia of these lymphoid areas, with the development of germ centres in the inflamed area in tissues where lymph nodes with germ centres are not found, is commonly enough seen and is strong evidence in support of Ribbert's views. Hyperplasia of the regional lymph nodes as a part of a local inflammatory process is a well-known occurrence. The writer accepts

Ribbert's interpretation of these lymphoid collections as rudimentary lymph nodes, and goes a step further. As he has repeatedly found lymph nodes with germ centres in newly formed inflammatory tissues on the pleura, peritoneum, and in peritoneal adhesions, etc., where a simple hyperplasia is out of the question, he believes also in a new formation of lymph nodes as a part of the inflammatory process, the new nodes arising from the proliferation of wandering lymphocytes. Inasmuch as the part played by the lymphocytes of the inflammatory infiltration is not phagocytic, it is but reasonable to believe that they must possess some other function. Further mention of this will be made later.

The cells of the large lymphocyte type may be in part derived from the small lymphocytes or, as is more probable, are for the chief part young formative cells derived from the connective-tissue cells and endothelium. It is impossible to decide morphologically. There are no staining methods by which young formative cells may be distinguished from lymphocytes. Transition forms appear to exist on both sides, and it is easily understood how some writers regard these cells as of connective-tissue origin, while others regard them as belonging to the white cells and coming from the blood-vessels.

The same difficulty of interpretation attends the plasma cells. These are round or oval cells staining deeply with methylene-blue and possessing excentrically placed nuclei, which have a chromatin network and five to eight chromatin granules. As the protoplasm is more compact toward the periphery, the nucleus appears to be surrounded by a lighter zone. Many writers regard these cells as a variety of lymphocyte and claim to have seen them emigrating from the blood-vessels. Others consider them to be of connective-tissue origin. As these cells show elongated forms gradually passing into cells taking part in the formation of scar tissue, and as the protoplasm of the plasma cell often shows an oxyphile hyaline change, the writer is inclined to accept the view of their connective-tissue origin, not upon the ground of a demonstrated histogenesis, but upon that of an apparent participation in connective-tissue formation. The plasma cells are particularly abundant in chronic infective inflammations, especially in those characterized by much scar-tissue formation. In blastomycetic dermatitis nearly every cell of the infiltration may be of the type of the plasma cell. Occasionally the plasma cells may act as phagocytes, the writer having seen them containing blastomycetes.

Multinuclear as well as mononuclear giant cells (Fig. 30) are also found at times in the cellular infiltration, particularly in chronic infective processes and about foreign bodies. In part they may be derived from leucocytes, but the majority are of connective-tissue or endothelial origin. Their function appears to be essentially protective.

Summing up, we may say that the cells of the inflammatory infiltration come in part from the blood, in part from the wandering cells of the tissues,

in part from hyperplastic or newly formed lymph nodes, and in part from the proliferation of connective-tissue and endothelial cells.

Phagocytosis.—The polymorphonuclear leucocytes and the derivatives of the fixed connective-tissue cells and endothelium have the power of taking up foreign bodies, particularly bacteria. To a less degree the cells of the large lymphocyte type show also the same property. To this process the term *phagocytosis* is applied; the cells exhibiting it are designated *phagocytes*. While such a property is, in the case of monocellular animals, chiefly a nutritive function rather than protective, it has evolved in the multicellular animals to a function of certain forms of cells serving chiefly as protective agents. Through chemotaxis the cells capable of phagocytosis are brought to the place where their function may be exerted to the greatest advantage. Particularly in the case of bacterial inflammations does this function serve the body as a protective factor. Great numbers of the bacteria are often taken up by the phagocytes and rendered inert or are destroyed within their protoplasm by means of intracellular chemical processes. Like the other protective factors of the inflammatory process, phagocytosis is not a perfect means of protection. The phagocytes themselves may be destroyed by the bacteria they have taken up, or they may fail to render inert those they contain within their protoplasm and may thus disseminate them throughout the body by means of the lymph or blood stream. In spite of these facts the unprejudiced observer must realize the great protective value to the body of phagocytosis as it ordinarily occurs in local pyogenic inflammations. The view that it is only an accidental utilization of a function primarily intended for nutrition is not in any way an argument against an appreciation of its utility.

Extracellular Protective Factors.—Not all the cells of the inflammatory infiltration have the power of phagocytosis, and the inference naturally arises that they must possess some other function as far as the inflammatory process is concerned. The fact that the blood serum and the serous exudate contain bactericidal substances (*alexins*) has been demonstrated by many investigators. The source of the alexins has been ascribed by many to the leucocytes, the antibodies arising either as a cell secretion or as a product of cell disintegration. That the leucocytes contain bactericidal substances has been shown by Buchner, Hankin, and Loewit. Organs and exudates rich in leucocytes yield bactericidal substances. Blood deprived of leucocytes shows a lessening of its bactericidal property. Further, Loewit has succeeded in extracting from washed leucocytes a bactericidal substance of great power.

According to Kanthack and Hardy, the eosinophile cells, which also respond to chemotactic influences but are not phagocytic, act protectively by discharging the eosinophile granules, which appear to affect the bacteria so that they are then taken up by the phagocytes. Stokes and Wegefarth hold that in the blood serum there are constantly present granules resembling those of the

eosinophile and neutrophile cells, and most probably derived from them. When these granules are filtered out of the blood it loses its power of destroying bacteria, but this property may be restored by adding leucocytes and granules. The researches of the last decade have made it evident that the cells of the leucocyte type afford the body protection not only by means of phagocytosis, but by producing and giving to the blood serum or inflammatory exudate bactericidal substances. Antitoxic substances may be produced in the same way.

Further, the leucocytes of the inflammatory infiltration may prevent the spread of a harmful agent by means of a densely packed cell barrier formed about the primary centre of injury. During the process of repair the white cells may also serve as a source of food to the formative cells, the latter often containing them in various stages of disintegration.

The Fluid Exudate.—That there is a direct relationship between the formation of the fluid exudate and the cause of the tissue lesion is shown by the fact that the former increases in amount and varies in character with the nature and severity of the irritation produced. The location of the injury and the general condition of the body and nervous system are also factors influencing the amount and character of the fluid exudate. As stated above, there are very good reasons for believing that the fluid exudate is not a filtrate pure and simple, but is a secretion of the vascular walls. Only by means of such a view is it possible to explain the character of the fluid, its difference in composition from the blood serum, and its varying character in different parts of the body. Such differences can be interpreted only as an indication of a selective activity on the part of the endothelium.

In the fluid exudate there are present both antibacterial and antitoxic substances, but, as mentioned above, these are most probably formed by the cells and set free into the fluid exudate. On a body surface the fluid exudate may wash away or dilute the injurious agent. In the tissue spaces the inflammatory œdema may also serve a similar purpose. The formation of fibrin in the exudate may also be a factor of advantage in limiting the spread of the bacteria. Since its production is associated with the disintegration of leucocytes and others of the wandering cells, it may be associated with the production of antibodies or ferments having the power of destruction or digestion of the irritant. Further, the fluid exudate may also serve in increasing the nutrition of the inflamed area and in this way promote cell growth.

Part Played by the Nervous System.—While acute inflammatory processes may occur independently of the central nervous system, the vessels of the inflamed area either responding directly to the irritant or through the peripheral nerves, it has been shown experimentally that removal of the vaso-constrictor influence accelerates an inflammatory reaction, while removal of the vaso-dilator influence retards it. Further, influences from the central nervous system alone, without the occurrence of a local injury, may set into action all

the phenomena of local inflammation. Such inflammatory reactions of nervous origin may be seen in cases of hysteria and in hypnosis. It is also probable that some of the so-called "sympathetic inflammations" are in reality referred processes having a central nervous origin. Inflammatory reactions may thus be produced along other branches of a nerve supplying a region in which there is a primary inflammation. Likewise, areas whose nervous supply comes from the same part of the brain or cord as that supplying an inflamed part may similarly be involved. Such questions, however, need more thorough investigations before we can unhesitatingly accept such a referred origin.

Of the existence of purely trophic nerves there is as yet not a shadow of absolute proof. The inflammatory changes so often seen in parts devoid of nervous supply, as the result of the section or complete destruction of the spinal cord or nerves supplying the part, may be explained as the result of the tendency of such tissues to receive trauma in connection with their lowered tone, due to the lack of exercise, disturbed circulation, etc.

Part Played by the Lymphatic System.—The important rôle played in the local inflammatory process by the rudimentary lymph nodes scattered throughout all tissues has been mentioned above. The larger regional lymph nodes and lymphatics have also an important part in inflammation, both clinically and pathologically. The lymph channels leading from the inflamed area become dilated as the result of the increased amount of lymph coming from the inflamed region. Since this lymph may contain the infective agent or chemical products formed by it, the endothelium of the lymphatics in question often becomes hypertrophic and may also exercise a phagocytic function. It is also an open question if such hypertrophic endothelial cells do not produce antibodies. There is also an increased number of leucocytes in the lymph coming from the inflamed part, and some of these may contain the injurious agent. Further, large numbers of red blood cells may be found in such lymph.

The lymph nodes receiving such lymph become enlarged and often painful. On section they are softer and moister than normal nodes, and usually are more or less congested. On microscopical examination they present a condition of congestion, œdema, and more or less hyperplasia of the lymphoid and endothelial elements. The lymph sinuses may at times be packed full of large hyaline cells of endothelial type. These especially are found to be acting as phagocytes, and contain red blood cells, blood pigment, leucocytes, or bacteria. Numerous mitoses are found throughout the lymphoid tissue. Leucocytes containing bacteria may also be found in the lymphoid areas as well as in the sinuses. That these bacteria are dead or are reduced in virulence is evident from the fact that in the great majority of cases they do not multiply or produce local cell necrosis. When the infective agent is of high virulence small areas of cell degeneration or necrosis are often found in the sinuses or lymphoid tissue, but the fact that the bacteria often soon die out in these areas without causing

further changes may also be taken as strong evidence of the protective functions exercised. Occasionally these are inadequate and the lymph nodes may become the seat of secondary inflammations, equalling or exceeding the primary in severity.

The blocking of the lymph sinuses through the proliferation and hyperplasia of the endothelial cells lining them serves as a barrier to the further spread of the infective agent, and gives opportunity for the exercise of the function of phagocytosis and the formation of antibodies. The large mononuclear cells (hyaline cells) lying in the meshes of the reticulum of the lymph sinuses often contain great numbers of disintegrating red blood cells and particles of blood pigment. They also take up disintegrating leucocytes. The number of eosinophile cells is usually increased in the regional lymph nodes during local inflammatory processes. They are most numerous at the borders of the lymphoid areas along the sinuses. The active proliferation of lymphocytes may also be interpreted as protective.

In the case of very virulent organisms or when the resistance of the body is lowered, the injurious agent may extend along the lymph vessels, exciting an inflammatory reaction along their course (*ascending lymphangitis*), and may also involve the nodes. The latter may present secondary foci without any signs of the intermediate involvement of the lymphatics. Primary cryptogenic infective inflammations are also not uncommon in lymph nodes. Such events simply go to show that the body's protective powers are not always adequate, and that at times the infective agent may conquer them and the process of tissue injury and reaction go on until the organism either finally overcomes or succumbs.

Reparative Factors.—As we have already seen, the slightest possible tissue lesion excites chiefly a formative reaction—the cells at or about the seat of injury divide and replace those killed by the injurious agent. In more extensive and severe tissue lesions, particularly those due to infective agents, the formative reaction may be longer delayed, owing to the progressive injury to the tissues about the growing colony of micro-organisms. Nevertheless, under all conditions cell proliferation is so constant a factor of the inflammatory reaction that it can be regarded not as a sequela alone, but as an essential part of the process. The tissue destroyed may be replaced by tissue of the same kind (*regeneration*) or by fibrous connective tissue (*repair, cicatrization*).

Regeneration (*Restitutio ad integrum*).—The process of regeneration is dependent upon the kind of tissue involved, the extent and severity of the lesion, and the mode of action of the injurious agent. If the cells of an inflamed area are but slightly damaged and retain their nuclei intact, they are quickly restored as soon as the injurious agent ceases to act. When single cells are lost without disturbance of the tissue organization as a whole, there occurs in the majority of tissues and organs a rapid replacement through the division

of the neighboring cells. The connective tissues, the epithelium of the skin and mucous membranes, liver and kidney, have the greatest regenerative capacity; while ganglion cells, bone cells, cartilage cells, and heart muscle have little or none. Cell proliferation may begin as early as eight hours after the lesion, but is usually recognizable at from twenty-four to forty-eight hours. Under certain conditions, particularly in the case of chronic inflammations, the process of regeneration goes beyond the degree necessary for the restoration of the part to its original state, and there result inflammatory overgrowths and hyperplasias, which may be of no service to the body or even of a disadvantage. Such hyperplasias are often found about the margins of slowly healing ulcers.

Repair.—When the tissue injury is so marked as to cause a break in the continuity of the mesodermal tissues, there is an incomplete regeneration in that there is formed at the seat of injury a new tissue, which differs more or less from the original tissue and shows also a more or less marked loss of function. This new tissue arises through the proliferation of the fixed connective-tissue cells and endothelium about the seat of injury. It consists essentially of proliferated connective-tissue cells (*fibroblasts*) and new capillary loops, and is infiltrated with mono- and polynuclear leucocytes. Formative cells arising from such tissues as the periosteum, bone-marrow, or muscle are known as *osteoblasts, chondroblasts,* and *sarcoblasts*, accordingly as they form bone, cartilage, or muscle. In the case of injury to epithelial tissue, newly formed epithelial cells may also be present in the formative tissue. The formative cells themselves have the power of amœboid movement and may appear as wandering cells. They are sometimes multinuclear or may appear as mononuclear giant cells. They vary greatly in form, are usually branched, and have large bright oval nuclei which do not stain deeply, thereby resembling the nuclei of epithelial cells. For that reason formative cells are often called *epithelioid* cells.

The formative tissue is known as granulation tissue. It is extremely vascular; hence to the naked eye it is red in color. The vessels (newly formed capillary loops) are characterized by a wide lumen and a wall consisting of a single layer of endothelial cells, which usually appear hypertrophic. The new capillaries arise as offshoots from the vessels in or near the inflamed area. The new capillaries extend out into the necrotic tissue or exudate at the seat of inflammation. Between them lie the fibroblasts, embedded in a fluid or semifluid intercellular substance infiltrated with leucocytes. As the fibroblasts increase in number and come to lie more closely together and to supersede the leucocytes, fine fibrillæ begin to appear in the intercellular substance. As this differentiation of the intercellular substance takes place, the formative cells become smaller, the blood-vessels contract, the granulation tissue gradually becomes changed into scar tissue, and the process of repair comes to an end. In the scar tissue small groups of lymphoid cells may remain for a long time

after all other signs of inflammation have disappeared. They may be interpreted as persistent hyperplastic rudimentary lymph nodes.

The phenomena of cell proliferation are essentially reparative, but they are also to some extent protective. The zone of granulation tissue about an abscess or at the bottom of an ulcer may act as a barrier against the further spread of the infective agent into the surrounding healthy tissues. The great number of thin-walled vessels with their active endothelial cells favors the production of a serous fluid containing antibodies. Afanassieff has shown that the action of the cells and serum of a healthy granulation tissue is bactericidal. Nevertheless, in some cases the formative tissue may become necrotic as the result of increased virulence of the infective agent, secondary or mixed infection, or weakened resistance due to circulatory disturbances, general anæmia, etc. Moreover, granulation tissue cannot restore the lost function of the part destroyed. Again, in some cases the formation of granulation tissue is so excessive as to be of disadvantage or even an injury to the organism, while in other cases still the resulting contraction of the scar tissue may cause additional functional disturbances. The reparative processes, like the protective, are also imperfect and inadequate.

6. Symptomatology.

The classical symptoms of the inflammatory process (*calor*, *rubor*, *dolor*, *tumor*, and *functio læsa*) may be manifested when the inflammation is located upon the surface of the body.

Calor (Heat).—The increased warmth of an inflamed area located upon the body surface is due to the active hyperæmia, the dilated blood-vessels favoring the greater amount of heat dispersion. The inflamed part becomes warmer than it was before only as the result of the increased blood flow. Although many writers have believed in a local increase of heat production, the most careful measurements have failed to demonstrate such a local rise in temperature, the temperature of the affected area never being higher than that of the blood in the left ventricle. The temperature of the body as a whole may be raised (fever). While measurements may fail to show any such increased local heat production, it is not at all improbable that such does occur as the result of molecular changes due to the action of the inflammatory agent. Such local increased heat production may easily be compensated by the local increase of heat dispersion, so that the local temperature may be maintained at no greater height than that of the internal organs. The increased temperature may serve a protective function in inhibiting or killing off the infective agent.

Rubor (Redness).—This is the direct result of the hyperæmia. At first, the inflamed area is uniformly bright red; later, as the blood current slows and a condition of stasis supervenes, the color becomes darker or even bluish, although around the periphery there may persist a bright-red zone. The central portion

of the damaged area may be anæmic and present a grayish or yellowish color, while around the periphery there is a surrounding zone bright red in color. As the new capillaries extend out into the necrosed area the latter gradually becomes red. During the course of the inflammatory process the diffuse red color often shows a mottling of darker spots, which do not become pale when pressed upon. They are the result of scattered hemorrhages.

Dolor (Pain).—The local pain and tenderness may be explained as the result of the increased pressure exerted upon the nerves, their direct irritation by chemical products, or their direct participation in the inflammatory process. In the case of inflammatory conditions of the internal organs, less often of the body surface, the pain is often referred to other parts. Usually this is to parts supplied by branches of the same nerve as that supplying the inflamed area.

Tumor (Swelling).—The swelling of the inflamed area is easily explained by the increased amount of blood in the part, the assemblage of cells, the increased formation of lymph, and the swelling of the tissue elements. The lymph stasis (inflammatory œdema) of the inflamed area plays a very prominent rôle in the enlargement of the inflamed tissues. It is due in part to the changes occurring in the regional lymphatics and lymph nodes, and in part to the diminished elasticity of the inflamed tissues.

Altered Function (Functio Læsa).—The alteration or loss of function of the inflamed part is the result chiefly of the tissue lesion, the injury or destruction of the parenchymatous cells, the result of pressure, etc.

Constitutional Symptoms.—Even in the simple, non-infective forms of inflammation there may be more or less constitutional disturbances, such as general malaise, headache, fever, etc., but these symptoms are exhibited to the most marked degree in the case of pyogenic inflammations. High fever, pain in the bones and joints, headache, gastro-intestinal disturbances, nervous disturbances, delirium, etc., may characterize the clinical course of these processes. These symptoms are due to the absorption of toxic substances (*ptomaïns* or *toxins*) from the inflammatory focus. To this general intoxication the terms *toxinæmia* or *septicæmia* are usually applied. The poisons absorbed in these conditions may cause a degeneration or necrosis of the epithelium of the liver or kidneys, and the symptoms of acute uræmia may be added to the others. In fatal pyogenic infections (*septicæmia* or *pyæmia*) the kidney lesion is usually the immediate cause of death (*acute degenerative nephritis*). The effects of such intoxications upon other organs—as the heart (*cardiac insufficiency, "heart failure"*), for example—may also be added to the general clinical picture. In the case of chronic inflammations a chronic intoxication may lead to secondary chronic inflammatory processes in the liver, kidney, or other internal organs. Chronic suppurative processes may give rise to a general *amyloid degeneration*, with its resulting clinical picture.

7. Involvement of the Organism in the Inflammatory Process.

The inflammatory process is essentially a local reaction to local injury. While the significance of the term might be extended to include the general processes of protection and defence (immunity) of the body against infections or intoxications, it is inadvisable to do so and better to reserve the term for the local reaction to injury. Further, in the case of general infection or intoxication of the body, the protective reaction is deprived of the greater part of the phenomena (hyperæmia, chemotaxis, emigration, serous exudation, etc.) that characterize the local reaction to injury. In the general reaction the chief phenomenon is the production of antibodies.

As a result of the local process, however, the general protective influences are often called into action, and, indeed, must be considered an essential part of the former. This is particularly the case when the inflammation is due to infective micro-organisms producing toxic substances, that when taken up into the blood and lymph cause injury to the cells of the body. Such poisons may arise from the growth of saprophytic micro-organisms in the necrotic tissue or in the exudates at the site of the primary lesion (*sapræmia*), or from toxins produced there by pathogenic organisms (*toxinæmia, septicæmia*).

The exciting cause (bacteria) may be spread through the body by means of the lymph or blood stream (*bacteriæmia*), and give rise to secondary foci of tissue injury and reaction (*lymphogenous* and *hæmatogenous metastasis*). In the case of pyogenic organisms the formation of secondary abscesses is known as *pyæmia*. The entrance into the blood of pathogenic micro-organisms and their growth there with the production of a general intoxication, but without the formation of secondary local inflammations, are usually called *septicæmia,* although this term is also applied to the condition of toxinæmia alone without the presence of bacteria in the blood. A combination of pyæmia and septicæmia may also occur (*septicopyæmia* and *pyosepthæmia*).

The general organism may also be affected in other ways by the local inflammatory process. In certain organs of the body auto-intoxications may be produced as the result of disturbed function due to inflammation. In the case of glands having an internal secretion, inflammatory processes may lead to severe or fatal disturbances of general metabolism. The interdependence of the function of one organ with that of another may also be disturbed as the result of local inflammation.

8. Classification of Inflammations.

According to their etiology inflammations may be classed in general as *traumatic, thermal, toxic, infective,* etc. The non-infective inflammations are often spoken of as *simple* or *aseptic.* The terms *idiopathic, sthenic,* and *asthenic* inflammations are no longer used.

Inflammations are classed as *acute, subacute,* and *chronic,* according to their course. When the process rapidly arises and quickly passes it is termed acute; when slowly progressive over a long period of time it is classed as chronic. Processes occupying an intermediate station may be known as subacute. In general, acute inflammations are characterized by abundant exudation, chronic inflammations by abundant production of connective tissue. Such a distinction does not, however, always hold good, but applies to the great majority of cases.

According to their location inflammations may be classed as *superficial, parenchymatous,* or *interstitial.* Superficial inflammations are those situated upon a body surface. Upon mucous membranes the inflammation is more often designated a *catarrh.* A parenchymatous inflammation is one characterized by degeneration or necrosis of the parenchymatous cells of an organ, while an interstitial inflammation is one in which the connective tissue or supporting stroma of the organ is chiefly involved. Such a distinction is somewhat artificial, since all inflammatory processes necessarily are interstitial, the interstitial reaction following and being dependent upon a damage to the parenchymatous cells. As a rule, the term parenchymatous inflammation is applied to conditions of tissue lesion (degeneration or necrosis) in which the inflammatory reaction may not yet have appeared, so that interstitial changes are not yet apparent. Since the term interstitial has largely come to be used as a synonym for *productive* inflammation,—that is, those inflammations which are characterized particularly by proliferation of the connective-tissue cells,—it serves to designate a certain class of inflammatory processes more or less chronic in nature and due chiefly to chronic intoxications.

The character of the tissue lesion also serves as a basis for a classification of inflammation. When tissue degenerations—such as cloudy swelling, hydropic, fatty, or mucoid degeneration—are more prominent as the tissue lesion than is the inflammatory reaction, the inflammation is usually styled *degenerative.* Since those forms which are characterized by mucoid degeneration usually occur upon mucous membranes, they are embraced in the more general designation of *catarrh.*

Tissue lesions of the nature of a marked necrosis give to the inflammatory process a *necrotic* character. The necrosis may be of the type of simple, coagulation, liquefaction, caseation, or gangrenous. A coagulation necrosis occurring upon a surface, particularly upon a mucous membrane, is usually styled *diphtheritic,* or the entire process may be known as *diphtheritis.* A fibrinous exudation is usually associated with the latter process. Secondary infection of the tissue lesion with saprophytic bacteria gives to the process the type of a *gangrenous* inflammation. A primary gangrenous inflammation may also occur. Infection with the gas-forming bacteria gives rise to an *emphysematous gangrene.* *Caseous* necrosis is found particularly in the case of certain chronic infective inflammations, tuberculosis, syphilis, etc.

The classification according to the character of the exudation serves a very practical purpose both clinically and pathologically. There may be distinguished, first, a *serous* inflammation, characterized by the production of a more or less abundant fluid exudate containing relatively but few cells. When the percentage of fibrin is so high as to make it a very prominent feature of the exudate, it is classed as *fibrinous*. When the cellular infiltration of the inflamed area is but slight, it is spoken of as a *small-celled infiltration;* but when the cells are so numerous as to pack densely the area, or when occurring in such numbers in the fluid exudate as to give it a thick, cloudy appearance, the inflammation is styled *purulent*. The liquefaction of the purulent area is known as *suppuration*, and the resulting mixture of leucocytes, tissue fragments, etc., is called *pus*. An exudate containing large numbers of red blood cells may be styled *hemorrhagic*. Various combinations of these forms may occur, such as *seropurulent, serofibrinous, fibrinopurulent,* etc.

Inflammations are also often classed according to their mode of resolution or the sequelæ of the inflammatory process. Some inflammations cause marked atrophy of the part affected (*atrophic inflammations*); others are characterized by marked proliferation of the connective-tissue stroma (*interstitial, productive, hyperplastic, indurative, cirrhotic,* etc.). Serous surfaces may become greatly thickened or opposing surfaces may become united through the organization of the exudates gluing them together (*adhesive* or *plastic inflammation*). In the case of certain chronic infections, large tumor-like growths of granulation tissue may be produced (*granuloma*). Marked sequelæ of such a nature as to characterize the inflammation occur more often as the result of protracted chronic inflammatory processes than as the result of the healing of acute processes.

Varieties of Inflammation.

Etiology.	Chronicity.	Location.	Tissue-Lesion.	Exudation.	Sequelæ.
Traumatic (simple). Thermal. Toxic. Infective.	Acute. Subacute. Chronic.	Superficial (catarrh). Parenchymatous. Interstitial.	Degenerative. Necrotic. 1. Simple. 2. Diphtheritic. 3. Liquefaction (suppuration). 4. Caseous. 5. Gangrenous.	Serous (catarrhal). Fibrinous. Purulent (suppurative). Hemorrhagic.	Atrophic. Hyperplastic (productive). Indurative. Adhesive. Granuloma.

9. Resolution of the Inflammatory Reaction.

That the inflammatory reaction be brought to a standstill and the process terminate in healing, it is necessary that the exciting cause should cease to act, the necrotic tissue and exudate be disposed of, the nutritive and circulatory conditions be restored to the normal, and the tissue defect, if any, be repaired.

Cessation of Cause.—The action of the injurious agent may be of very

short duration, as in the case of certain forms of trauma. In the case of poisons the pouring out of a fluid exudate may dilute the irritant or wash it away. In infective inflammations the bacteria may be washed away by the fluid exudate, cast off with tissue sloughs, or rendered non-virulent, or are killed by the phagocytes and the extracellular bactericidal substances formed during the inflammatory reaction. The irritant substances formed by bacteria may be neutralized by antitoxins.

Disposal of Necrotic Tissue.—Dead cells or tissue elements may be cast off as a slough, liquefied, in part absorbed and utilized as nutriment, or replaced by granulation tissue (organization). The sequestration and absorption of necrotic tissue require a certain amount of time. The greater the amount of dead tissue, the slower the removal and the more protracted the healing process. As a rule, the inflammation persists as long as necrotic tissue is present.

Disposal of the Exudate.—Serous exudates may be quickly taken up by the lymph stream. Fibrinous exudates, when soon liquefying, may also be quickly absorbed. Firm fibrinous exudates and large collections of pus are removed with difficulty and prolong the course of the inflammation. Exudates upon a surface may be cast off. Firm exudates often become liquefied and are then more easily absorbed. When liquefaction does not take place, the exudate acts as a foreign body and prolongs the inflammation, eventually becoming replaced by granulation tissue (*organization*). Scar tissue is ultimately formed.

Restoration of the Normal Circulation.—With the cessation of the action of the injurious agent and the removal of dead tissue and exudates and the establishment of reparative processes, the hyperæmia of the vessels of the inflamed area subsides, the leucocytosis and emigration diminish, and the number of wandering cells in the tissue spaces becomes lessened. Ultimately the normal vascular conditions are restored, and the nutrition of the part again becomes normal. When the irritant is of slight intensity and duration, as in the case of a slight trauma, burn, corrosive poison, etc., the restoration of the vessels may take place in a very short time. In the case of more extensive tissue lesions, the normal vascular conditions are more slowly restored.

When granulation tissue has replaced a tissue defect, the newly formed blood-vessels become smaller as the transformation into scar tissue takes place. Collections of lymphoid cells may persist long after all other signs of the inflammatory reaction have disappeared, and are best explained on the ground of hyperplastic rudimentary lymph nodes. In general, the restoration of the normal vascular conditions depends upon the duration of the exciting cause, the amount of dead tissue and exudate to be disposed of, and the size of the defect to be filled in.

Repair of the Tissue Defect.—This is accomplished by cell proliferation leading to regeneration or repair, according to the nature and severity of the inflammatory agent and the character of the tissue involved. When only single

cells are lost or the organization of the tissue is but slightly disturbed, there occurs in the majority of tissues a rapid regeneration. In the case of more extensive lesions, solution of continuity, wounds, fractures, necrotic inflammations, suppurations, etc., there is first formed a granulation tissue, which later becomes changed to scar tissue. Evidences of the beginning of cell proliferation may be seen microscopically as early as eight hours after the injury, but the process is usually not well established until after twenty-four hours or later. The establishment of cell proliferation is dependent upon the cessation of the exciting cause and the supply in abundance of the materials necessary for the nutrition of the cells. In some cases the phenomena of cell proliferation are so early established and form so marked a characteristic of the inflammation as a whole that it may be styled a productive or a proliferating inflammation. All factors delaying the progress of healing, such as large areas of necrosis, large masses of purulent or fibrinous exudate, etc., give to the inflammation the character of a chronic process.

10. The Healing of Inflammations.

The termination of the inflammatory reaction is known as *healing.* The factors constituting the resolution of the inflammatory process and upon which the healing process depends have been considered in the previous section. It is, of course, evident, according to the standpoint taken in this article, that the entire course of an inflammation is directed toward a common end—that of healing; and it is, therefore, not possible to separate wholly the reparative factors from those purely protective. While the entire inflammatory reaction is, broadly viewed, *a healing process*, the term is used here in a narrower sense as applying to the final phases of the reaction.

Healing by First Intention (*per primam intentionem*).—In the case of an incised wound of the skin whose edges are glued together by serum or blood or are held together by sutures, the inflammatory reaction, in the absence of bacterial infection, is slight. Along the edges of the wound there is at first an abundant exudation of serum containing more or less blood; this, coagulating, holds the opposing wound surfaces together. At the same time there occurs along the edges of the wound a cellular infiltration, which is usually not very marked and reaches its maximum in a few days. When sutures have been used the infiltration is usually more marked about these than along the edges of the wound. By the end of twenty-four hours cell proliferation is usually well established along the edges of the wound. By the third or fourth day there is formed a granulation tissue, which replaces the exudate or blood clot between the wound surfaces and unites them together. The formative tissue extends also on both sides of the wound for some little distance along the blood- and lymph-vessels into the neighboring sound tissue, thus blending the wound into the neighboring tissue in such a way that the edges of the original line of

incision become indistinct. (See Fig. 31.) At the same time a regeneration of the surface epithelium is taking place, the epithelial cells at the edge of the wound pushing over the wound surface and dividing to form many layers. The formative tissue along the line of the incision gradually becomes less rich in cells, its blood-vessels contract, and there is a differentiation of fibrillæ in its intercellular substance. Nevertheless, for a long time afterward the scar thus formed may show evidences of proliferation and cellular infiltration. Finally, the place of the incision can no longer be made out, as the line of scar tissue comes to re-

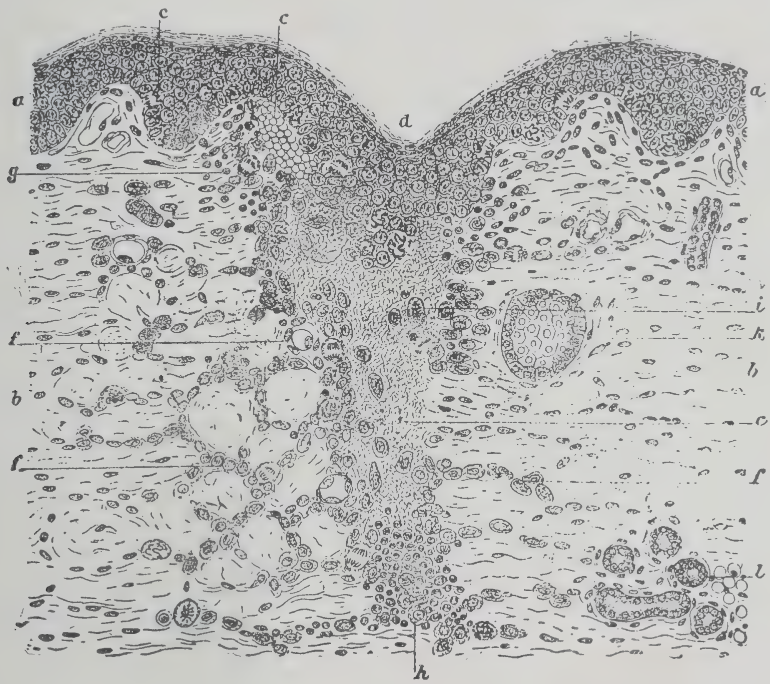

FIG. 31.—Healing of Incised Wound of Skin united by Suture (Flemming's solution, safranin). Preparation made on the sixth day. *a*, Epidermis; *b*, corium; *c*, fibrinous exudate, in part hemorrhagic; *d*, newly formed epidermis, containing numerous division figures, and with plugs of epithelium extending into the underlying exudate; *e*, division figures in epithelium at a distance from the cut; *f*, proliferating embryonic tissue, developing from the connective-tissue spaces, and containing cells with nuclear division figures, and in part also vessels with proliferating walls; *g*, proliferating embryonic tissue with leucocytes; *h*, focus of leucocytes in deepest angle of wound; *i*, fibroblasts lying within the exudate, one showing a nuclear division figure; *k*, sebaceous gland; *l*, sweat gland. × 70. (*After Ziegler.*)

semble the neighboring connective tissue. The time necessary for the complete healing of such a wound depends upon its size, the thickness of the layer of exudate or blood clot lying between the opposing wound surfaces, and the proliferative capacity of the tissue. The formation of granulation tissue along the line of incision is not always uniform; it may be absent in places or vary greatly in amount at different levels. The surface epithelium may extend across the wound before the formative tissue has developed below. Occasionally it may become hyperplastic. When much scar tissue is formed, its later contraction

causes a flattening or even depression of the cutaneous surface. The papillary bodies may not be regenerated and a smooth scar may result. (See Fig. 32.)

Healing by Second Intention (*per secundam intentionem*).—In the case of an open wound of the skin whose edges cannot be brought together, there occurs, in case the wound does not become infected, a serous or bloody exudation followed by cell proliferation at the base of the wound. Within twenty-four hours the base of the wound is deep red in color and more or less swollen. It is covered with a reddish-yellow exudate. After twenty-four hours there begin to develop over the base of the wound small red papules of formative

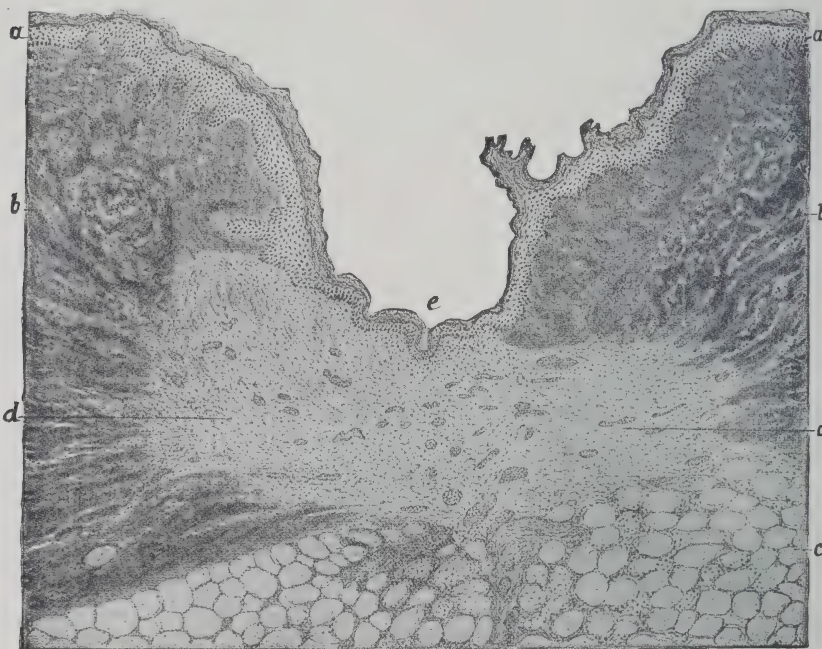

FIG. 32.—Cutaneous Portion of a Laparotomy Cicatrix, Sixteen Days after the Operation (Mueller's fluid, hæmatoxylin, Van Gieson's). *a*, Epithelium; *b*, corium; *c*, subcutaneous fat tissue; *d*, scar in corium; *e*, new epithelial covering; *f*, scar in fat tissue. × 38. (*After Ziegler.*)

tissue. These increase in number and become confluent, so that by the fourth or fifth day the entire floor of the wound may be covered by a granular red surface, over which lies a more or less thick, grayish, gelatinous layer of exudate. This exudate is very rich in albumin and fibrinogen, and contains many round cells, chiefly of the polymorphonuclear variety ("*pus cells*"). Many of these show degenerating nuclei.

The formative tissue (*granulation tissue*) at the base of the wound consists of fibroblasts, newly formed capillary loops, and leucocytes embedded in a fluid or semifluid intercellular substance. In the latter there is soon developed a fibrillar ground substance. Over the surface of the granulation tissue there is a layer of exudate rich in fibrin and containing many pus cells.

(See Fig. 33.) At the edges of the wound there is a rapid proliferation of the epithelium, and a layer of newly formed epithelial cells pushes in from the periphery over the wound granulations, often extending deep down underneath the superficial layer of exudate. As the wound becomes covered with epithelium and as the granulation tissue is gradually differentiated into fibrous connective tissue, the proliferative processes gradually come to a standstill. The scar thus formed is at first very vascular and of a red color. It is often elevated and covered with hyperplastic epithelium. As the scar tissue contracts the vessels become smaller and many of them are obliterated. The new tissue also loses in volume. Ultimately the scar becomes pale, smooth, and often depressed. The papillary bodies are either not regenerated or they are reproduced only to a slight degree. For a long time the tissue of the scar is rich in cells, but these gradually become reduced in numbers and the tissue becomes dense and hyaline, showing relatively few cells. New elastic fibres may be formed. The regeneration of the cutaneous glands depends upon the severity of the original injury. If portions of the glands are preserved, new glands may be formed from these.

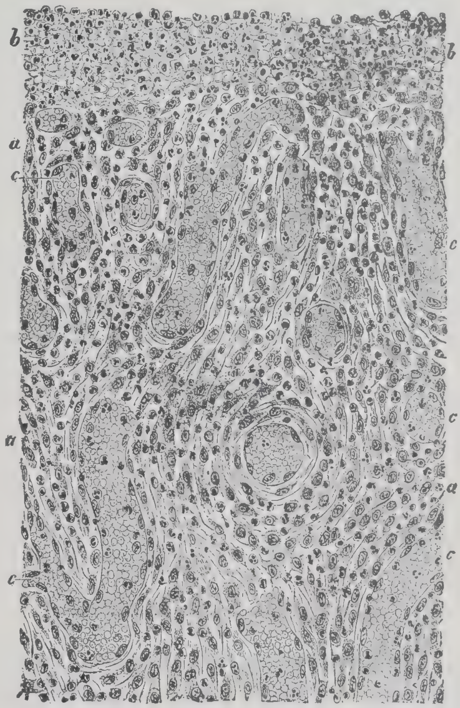

Fig. 33.—Wound Granulations from an Open Wound with Fibrinopurulent Covering (Mueller's fluid, hæmatoxylin). *a*, Granulation tissue; *b*, fibrinopurulent layer; *c*, blood-vessels. × 135. (*After Ziegler.*)

The process of healing by second intention is the same in the case of wounds of the internal organs in which the defect is large enough to be filled in with granulation tissue visible to the naked eye. In the case of surfaces not covered with epithelium, the new scar tissue is covered with endothelium (mesothelium) or becomes adherent to neighboring structures. In the case of infected wounds and in ulcers due to pathogenic micro-organisms, the process of healing is essentially the same, although more prolonged. In the case of large defects healed by second intention, the resulting contraction of the scar tissue may lead to marked deformities of the organ involved.

Healing of Abscesses.—In the case of small abscesses the pus may be quickly liquefied and absorbed, and the defect is closed up by granulation tissue which is transformed into scar tissue. In the case of larger abscesses there is formed about the periphery of the cavity a zone of granulation tissue known

as the abscess membrane (*pyogenetic membrane*). As the contents of the cavity are gradually liquefied and absorbed, the zone of granulation tissue extends toward the centre and gradually fills up the defect. Scar tissue is then formed and the process of healing is complete. If the process of liquefaction and absorption is incomplete, the pus may become inspissated or calcified.

Healing of Ulcers.—Ulcers heal by second intention, as described above.

Healing of Empyemata.—Large amounts of pus may be absorbed from the body cavities. When the process of absorption is slow or if the pus is inspissated, the tissues enclosing the pus produce formative tissue and the healing process is precisely the same as that in the case of an abscess. Large amounts of granulation tissue may be formed in the case of chronic empyemata. If the process is incomplete the remains of the pus may become calcified.

Healing of Fibrinous Inflammations.—Upon a mucous surface fibrinous exudates are cast off or liquefied. Only rarely (healing diphtheritic processes) do they become organized. In the case of the pulmonary air spaces, masses of

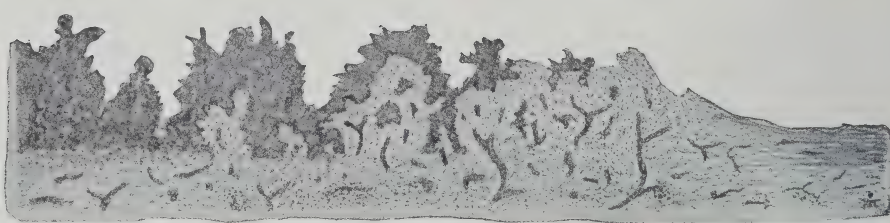

FIG. 34.—Scheme of the Organization of a Fibrinous Exudate on Serous Membrane. 1, Cellular infiltration beneath the fibrin; 2, first extension of fibroblastic tissue into the fibrin; 3, replacement of fibrin by fibroblastic tissue; 4, fibrin nearly wholly replaced by vascular formative tissue; 5, complete replacement of fibrin and beginning contraction; 6, contraction and transformation into scar tissue. (*After Ribbert.*)

coagulated exudate when not liquefied and absorbed may be replaced by granulation tissue, which fills up the alveolar spaces, leading to an induration of the lung (*fibroid pneumonia*). The fibroblastic proliferation proceeds either from the connective tissue of the septa or from that of the alveolar walls.

Upon serous surfaces a deposit of fibrin usually leads quickly to proliferative processes, so that as early as the fourth day fibroblasts may be seen extending up into the fibrinous layer. This is soon followed by a growth of capillary loops, and the fibrin layer is gradually replaced by a vascular granulation tissue, which later becomes changed to a dense hyaline scar tissue. (See Fig. 34.) Remains of the fibrin may persist in the new tissue for a long time. If the exudate was limited in area the new tissue becomes covered with endothelium (mesothelium), but when it is of large extent the opposing serous surfaces usually become united by the process of organization (*adhesions*). Such inflammations are usually spoken of as *adhesive*. (See Fig. 35.) It is evident that the character of the healing process is dependent upon the amount of the fibrinous exudate and the

situation and relations of the affected serous surface. In the case of small deposits the organization of the fibrin leads to thickenings of the serous membrane. The gluing together of two serous surfaces by a fibrinous exudate leads also to the formation of adhesions. When the amount of fibrin is small and the two surfaces move upon each other, stringy adhesions may be formed. Large amounts of fibrin may fail of absorption and become inspissated or calcified.

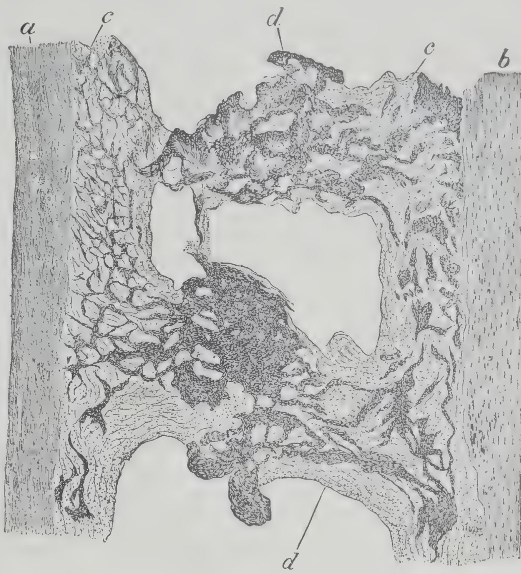

Fig. 35.—Formation of Adhesions between the Layers of the Pericardium in Fibrinous Pericarditis. *a*, Epicardium; *b*, parietal layer; *c*, formative tissue, containing remains of fibrin; *d*, connecting bridges of formative tissue. (*After Weichselbaum.*)

Healing of Thrombi.—Coagulated masses in the blood-vessels are replaced by connective tissue in the same manner as are fibrinous exudates upon serous surfaces. There is a fibroblastic proliferation of the cells in the vessel wall. Fibroblasts and newly formed capillary loops extend out into the thrombus, which is gradually replaced by vascular granulation tissue, which later is transformed into a denser fibrous connective tissue. The vessel lumen may be obliterated or the vessel wall may present local thickenings. Failure of organization may be followed by calcification of the thrombus.

Healing of Necrotic Areas.—Masses of necrotic tissue that cannot be disposed of by sloughing and sequestration are replaced by formative tissue and scar tissue, in the same manner as takes place in the case of fibrinous exudates and thrombi. The organization begins at the periphery and extends toward the centre of the dead area. Large masses may become encapsulated, the central necrotic material becoming inspissated, calcified, or liquefied.

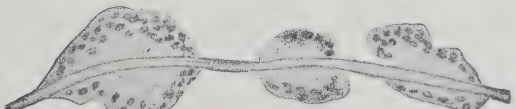

Fig. 36.—Foreign-body Giant Cells attached to Silk Thread. (*After Ribbert.*)

Healing of Foreign Bodies.—In the case of bland foreign bodies that are easily absorbed, the process of healing takes place in the same manner as in the case of fibrinous exudates or necrosed tissue. The foreign substance is

liquefied and the defect replaced by scar tissue. Small bodies that cannot be liquefied are taken up by phagocytes and in the course of time are gradually removed from the body (dust, carbon, tattoo, etc.). In the case of bodies too large to be taken up entire by phagocytes, there occurs in the granulation tissue developing about them a formation of large multinuclear giant cells, which attach themselves to the surface of the foreign body and cling to it (*foreign-body giant cells.*) (See Fig. 36.) These cells resemble the physiological osteoclasts. If the foreign body is slowly soluble (catgut, etc.) these cells gradually bring about its disintegration. When the foreign body contains crevices, the protoplasmic processes of the giant cells extend into these and gradually widen them. If the foreign substance is insoluble (hairs, silk, silver wire, etc.), the giant cells covering it gradually give place to a capsule of scar tissue (*encapsulation*). In the case of smooth bodies (glass, etc.) the amount of granulation tissue formed

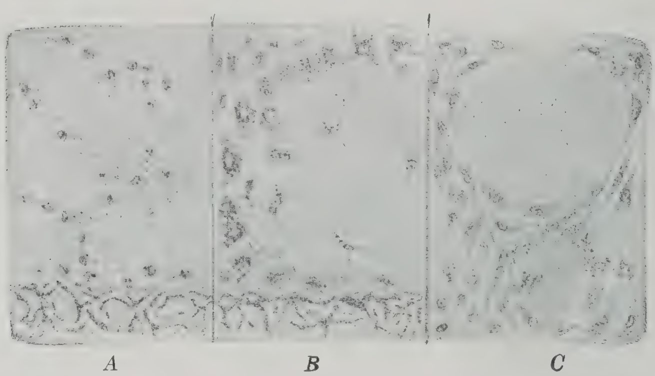

FIG. 37.—Anterior Chamber of Rabbit's Eye after Injection of Agar. *A*, Three days later. Iris below; above, the homogeneous agar containing leucocytes and some fibrin. *B*, eight days later; large formative cells about the agar mass. *C*, three weeks later; spindle cells replacing agar. (*After Ribbert.*)

may be very small. Not all foreign bodies excite the production of giant cells; hard bodies coming from without the body favor most their production, but extrinsic soft bodies may also cause their formation in large numbers. (See Figs. 37 and 38.) They are, however, frequently seen about necrotic tissue (dead muscle fibres). The giant cells arise from the connective-tissue cells and the endothelium. Mitotic division of the nuclei takes place without division of the protoplasm. While many of the giant cells appear to undergo disintegration, some split up into fibroblasts and take part in the production of scar tissue.

The leucocytes also play an important rôle in the reaction against foreign bodies. They usually quickly assemble at their site, take them up when possible, penetrate into their crevices, and aid in the process of disintegration. Fibroblastic proliferation follows or is associated with the assemblage of leucocytes, many of the fibroblasts acting also as phagocytes.

11. Sequelæ of Inflammations.

The sequelæ of inflammations depend upon the nature of the exciting cause, the location of the tissue involved, its character and condition, the severity of the process, the length of the course, and the nature of the healing process. In a general way the most important sequelæ of inflammation are those dependent upon cicatrization. The formation of scar tissue with subsequent contraction may lead to extensive atrophy of the parenchyma of the affected organ. The obliteration of serous cavities or the formation of adhesions on serous membranes may lead to serious impairment of function. The constriction of the

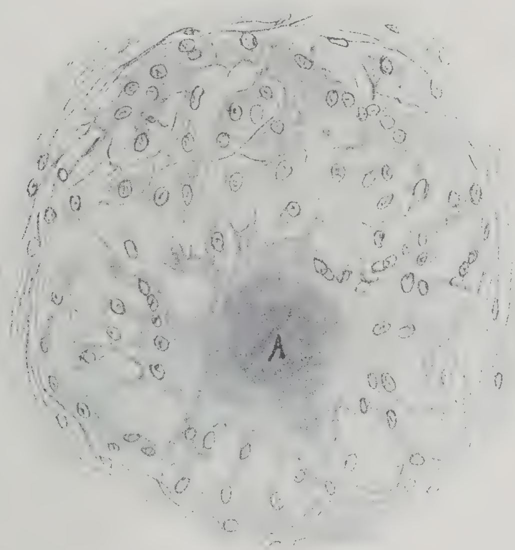

Fig. 38.—Subcutaneous Injection of Agar. Ten weeks after. *A*, Agar mass in centre of large, multinucleated giant cell showing numerous processes. (*After Ribbert.*)

intestine by such bands of inflammatory adhesions may lead to fatal results. The contraction of scar tissue leads also to surface disfigurations and deformities. The involvement of old nerve trunks or of newly formed nerve fibres in the scar tissue may give rise to an "*irritable scar.*" Through the organization of fibrinous exudate upon their serous surfaces the capsule of such organs as the liver, spleen, etc., may be greatly thickened. This thickening of the capsule may lead to a further secondary atrophy of the parenchyma. The obliteration of blood-vessels through the organization of thrombi or connective-tissue proliferation of their walls leads likewise to secondary parenchymatous atrophy. Through the organization of exudates lying within small body cavities, as the pulmonary air spaces, induration of the affected organ follows and the spaces are wholly or partly obliterated. The contraction of the scar tissue following the healing of ulcers of body passages, such as the œsophagus, stomach, intes-

tine, etc., may result in partial or complete stenosis. Inflammatory atresias may occur (vagina).

On the other hand, many inflammatory processes lead to an overproduction of tissue. Extensive new formation of bone may follow chronic inflammations of the periosteum or bone marrow. Connective-tissue hyperplasias of great extent may be associated with the presence of filaria in the lymphatics of the scrotum and extremities (*elephantiasis*). Chronic inflammations of the skin and mucous membranes may be followed by polypoid or wart-like hyperplasias of the connective tissue and epithelium (*condylomata*). Similar hyperplasias may be seen about the edge of chronic ulcers and in association with chronic infective processes. Many chronic infective inflammations are characterized by tumor-like formations of granulation tissue (syphilis, tuberculosis, leprosy, etc.). These are usually classed under the head of *infective granulomata*. The process of regeneration often leads to an overproduction of epithelial structures, as happens in the new formation of bile ducts occurring in hepatic cirrhosis.

12. Significance of the Inflammatory Process.

Summing up the features of the inflammatory reaction, we find that the process as a whole is of decided advantage to the organism. Chemotaxis, phagocytosis, increased formation of lymph, hyperæmia, emigration of white cells, formation of extracellular and intracellular bactericidal substances, production of antitoxins, formation of cell barriers, the increased temperature, cell proliferation, etc.—all of these factors in the process can be interpreted as serving for the protection or repair of the organism. Inflammation may then be considered a body function. As a matter of fact, practically all the factors of the inflammatory reaction are constantly active in the body. Phagocytosis, chemotaxis, lymph formation, cellular emigration, cell proliferation, etc., are constantly occurring in the body, but become manifested as inflammation only when occurring in a greater degree than under ordinary conditions. Like all other body functions, that of inflammation is subject to disturbances and is often very imperfectly carried out. When the great variety of factors and conditions influencing it is considered, there need be no wonder at the fact that in the struggle for protection and defence the inflammatory function as a whole may appear as a harmful process rather than as a preservative one. The inflammatory factors work more or less blindly, without discrimination. The inflammatory reaction cannot adapt itself to the anatomical and functional peculiarities of different organs and tissues. Thus an inflammatory reaction that may be successful from the standpoint of protection in other parts of the body may lead to fatal results in the larynx or brain, as the result of occlusion or pressure. Welch speaks of the "excesses, disorders, and failures incident to inflammation." As the adaptation of the animal body to the extrinsic factors

influencing it is an imperfect one, so is the local reaction limited and imperfect. Against some injurious agents the organism is able to produce but a slight reaction. The serous exudate may have little or no bactericidal action. The formation of antitoxin may be limited or not occur. Positive chemotaxis may not occur. The phagocytes, instead of destroying, may be destroyed or may transport the injurious agent (bacteria) to other parts of the body. The pouring out of an exudate into the pericardial cavity may seriously impair the efficiency of the heart. Collections of exudate in the pleural cavity cause interference with both respiratory and cardiac functions. Meningeal exudates cause cerebral compression; the filling up of the pulmonary air spaces with exudates causes respiratory and circulatory embarrassment; inflammatory processes upon the heart valves are followed often by stenosis or insufficiency, etc. The contraction of scar tissue may lead to severe secondary anatomical changes and functional disturbances. In its somewhat blind method of carrying out its protective function, the inflammatory reaction creates conditions that are in themselves harmful or dangerous. In the attempt to overcome the primary injury, the creation of such dangerous conditions is at times unavoidable.

While such injurious effects of the inflammatory reaction give to it, when viewed from the standpoint of the clinician, the character of a harmful process demanding surgical intervention, they should not blind him to the essential biologic facts. *Inflammation is an exaggeration of normal body functions—a struggle for protection and self-preservation—becoming manifest as the reaction to local injury.*

13. General Indications for Treatment of Inflammation.

While taking the ground that the inflammatory reaction is but an exaggeration of normal body functions aimed at protection and repair, the necessity for medical and surgical intervention is not denied. On the contrary, such active intervention becomes more clearly indicated as the limitations and imperfections of the protective and reparative processes are recognized and the better understood. Having gained the knowledge that inflammatory processes possess a unity, we are put in a position to apply logical and scientific methods of treatment. And it is to this knowledge that the surgery of the last twenty years owes its wonderful advance and its brilliant victories. The discovery of the nature of the most common and important etiological factors of inflammation, and the knowledge of the body's means of defence and protection against these agents, have raised the treatment of inflammation to a wonderful plane of advance. Surgery, in so far as the treatment of inflammation is concerned, has, also, become protective and defensive. Realizing the limitations and imperfections of the body function, it attempts to aid its protective and defensive powers, to limit them when necessary, and to avoid the "excesses, disorders, and failures"

of the inflammatory reaction. The surgeon supplies the additional higher function of judgment and discrimination in the struggle for self-preservation. He seeks to avoid the most dangerous etiological agents, to prevent simple injuries from becoming progressive (infection), to limit infection when it has occurred, to aid in the destruction of the infective agent, to control the body's defensive powers in such a manner that damage may not result from the collection of exudates, to shorten the course of the reaction by the removal of necrotic tissue, exudates, etc., and to further the course of repair by the coaptation of wound surfaces, removal of dead tissue, etc.

In addition, the surgeon endeavors to support the organism as a whole, to keep up its tone, to increase its resistance, to counteract the effect of general intoxications, and to prevent their occurrence. By means of bactericidal substances and antitoxins the body may be rendered immune to certain agents that otherwise would excite tissue lesions and inflammatory reaction. The surgeon may supply at the very beginning, before serious damage is done, those elements which the body itself can produce only later in the course of an infection after more or less severe local injury has occurred. To this especial branch of treatment we look for greater results.

The aid which the surgeon brings to the body's protective forces is, however, still imperfect. The course of many infections he cannot check, his operative procedures may often do more harm than good, the bactericidal substances which he uses may injure the tissues, he may lower the local resistance instead of raising it by the removal of exudates, and he may be the means of dissemination of the infective agent. Such imperfections of surgical technique we look for the future to remove. As our knowledge of the biologic facts underlying the inflammatory reaction increases, so will the methods of treatment become perfected.

II. ACUTE INFLAMMATION.

1. Acute Simple Inflammation.

The term simple inflammation is usually employed to indicate a reaction to trauma, or to thermal or chemical agents, rather than one resulting from infection (infective inflammation). The term "*aseptic inflammation*" is sometimes used as a synonym, but the impropriety of this is evident. Since the non-infective inflammations are usually non-exudative in character, these terms have also been used interchangeably. Further, since the non-infective inflammations usually terminate in repair or regeneration, they are often spoken of as *formative inflammations*. But inflammatory reactions of exactly the same clinical character may be produced by bacteria of low virulence, the colony quickly dying out, so that the process is non-exudative and non-progressive. Bacterial products (toxins) produce similar inflammatory reactions. For that reason it is not

best to limit the term to non-infective processes, even though in the great majority of cases simple non-exudative inflammations are not the result of infection. The term simple inflammation is, therefore, used here to indicate the simple reaction to injury when it is characterized not by exudative processes but by formative.

Simple inflammations may be caused by mechanical injury (friction, blows, cuts, wounds of any nature, surgical operations, etc.), burns, freezing, corrosive poisons, poisons having local irritant action, electricity, radioactivity, anæmia, disturbed nutrition, etc. As mentioned above, an infection with germs of low virulence that quickly die out may produce a similar clinical and histological picture.

The symptoms of simple inflammatory reactions are the five cardinal ones, more or less modified by the etiological factor and the location of the injury. As a rule, the hyperæmia of simple inflammation is less marked than in the exudative forms. Since the various forms of simple traumatic injury are the most common causes of simple inflammation, the symptoms usually observed are, first, a certain degree of redness, warmth, and swelling about the injury, with more or less pain or soreness. The intensity and character of the pain are dependent largely upon the location of the lesion. Even with the slightest wound there may be some constitutional symptoms, notably some elevation of temperature. The general symptoms are usually not marked unless the wound is severe or covers a large surface. Symptoms of shock may be associated with those directly due to the injury. As simple inflammations are not progressive and do not spread, the symptoms usually reach their height during the first two or three days and then gradually disappear as the process undergoes resolution.

The microscopical picture is that of a simple inflammatory reaction characterized chiefly by the assemblage of wandering cells and the proliferation of the connective-tissue cells and endothelium. The healing of wounds may take place either by first or by second intention, according to the character of the wound.

Treatment.—The treatment of simple inflammations is directed chiefly toward the prevention of infection with pyogenic organisms, to minimize the tissue damage as much as possible, and to hasten resolution. At the same time attention is directed toward the relief of the symptoms.

General Measures.—Of the more general measures to be carried out in the treatment of simple inflammations, rest is of the greatest importance. If there are constitutional symptoms (fever) the patient should be kept in bed; otherwise, rest of the affected part will suffice. Elevation of the inflamed part often gives marked relief. The diet is of great importance, particularly after surgical operations. Nutritious and easily digestible food should be supplied. Milk diet, either as fresh or sterilized milk, or mixed with lime water or potassium bicarbonate, is indicated after most operations, except those in which move-

ments of the bowels are not desired for several days (operations upon rectum and perineum). Light nutritious diet may be substituted for the milk diet or may follow it, as indicated. Boiled or distilled water should be given frequently in small quantities. Nutrient enemata may be given when food cannot be borne by the stomach. Thirst is often markedly relieved by enemata of physiological salt solution. Several days after the operation a more varied solid diet may be given. Alcohol should be used with caution. It may be of service as a temporary stimulant when the pulse shows signs of cardiac weakness. During convalescence the lighter alcoholic drinks may be used as tonics. On the whole, the general treatment is directed toward the support and building up of the organism.

Of actual drug treatment little or none is usually indicated. Antipyretics probably do actual harm and are contraindicated in surgical cases. Purgatives are to be used with judgment and discrimination for the purpose of preventing intestinal auto-intoxications. They are also occasionally indicated to relieve the tension arising from collateral congestions and to excite peristalsis. Diaphoretics and diuretics are now rarely employed. Pure water given frequently in small quantities serves best for these indications. When the pain is severe morphine may be used with discrimination, but it should never be given as a matter of routine. The first night after an operation may often be passed to greater advantage if a hypodermic injection of morphine is given. Under other conditions one-eighth to one-sixth of a grain by the mouth usually suffices. If indicated it may be given in a rectal suppository. On the whole, it is better to look upon the use of morphine as a last resort and to avoid its use in private practice whenever possible. The pain and restlessness may often be successfully combated by other means. Warm baths, hot or cold pack, potassium bromide, sulfonal, trional, and other hypnotics may be used. Phenacetin is often of service in relieving neuralgic pains. Chloral should be used with the same precautions as morphine. The period of convalescence from injury or operation demands careful hygienic measures. Massage, directed active and passive exercise, proper diet, rest, etc., should be systematically carried out according to the needs of the case. Tonics may be given when the patient is anæmic or shows little inclination for food.

The symptoms of *shock* are often added to those of the inflammatory reaction. This is most likely to be the case after injuries to the abdominal cavity, scrotum, spinal column, etc. Extensive burns of the skin, lightning or electrical shocks, corrosions, etc., are frequently followed by shock. Conditions of nervous excitement predispose to shock, while narcosis or alcoholic intoxication serves to inhibit it. The treatment will be considered under the proper heading.

Local Treatment.—The chief consideration in the local treatment of injuries is the prevention of infection. The general principles of aseptic surgery will be treated in another chapter. In this place only the treatment of the

simple inflammatory reaction following the proper treatment of the wound or injury will be considered. It will be assumed that the wound or part has been carefully cleaned and made aseptic, hemorrhage checked, coaptation secured, and proper dressings applied. Under such conditions in the great majority of cases no especial local treatment will be found necessary. Attention to the general principles given above are usually sufficient, inasmuch as the inflammatory process quickly reaches its height and passes to a rapid resolution. Nevertheless, there are certain general principles of local treatment which may often be applied with the greatest benefit. These principles all aim toward the relief of unpleasant symptoms and to assist the resolution of the process.

Rest of the affected part is one of the most important factors in relieving the pain, reducing the amount of tissue damage, and preventing unnecessary exudation and swelling. The position of the part is also of great importance in assisting the reaction. As a rule, elevation of the injured part is found to be of advantage. Strapping, bandaging, or encasing the part in plaster-of-Paris dressings may be necessary to secure immovability. In the case of fractures splints are used. Functional rest of the injured part is often desirable or necessary, in order to bring about resolution. This may be secured in various ways, according to the organ affected.

Cold is often of great service in reducing the discomforts of the inflammation. Applied by means of the ice bag or ice coil, through which a constant stream of ice water is kept flowing, it often greatly reduces the pain and lessens the exudation and swelling. Its use is contraindicated when the vitality of the part is lowered and when the venous stasis is marked. When the temperature of the inflamed area is not much increased, its color cyanotic, and the swelling marked, the application of cold may cause actual damage. Gangrene may follow the over-zealous use of the ice bag. The simple application of cold cloths or the immersion of the affected part in cold water often has a marked soothing effect. Cold water allowed to drip constantly upon the bandage or dressings may be employed for the same purpose. The individual conditions of the case are, of course, to be considered in the employment of cold according to any one of these methods.

Heat is also an important factor in the treatment of simple inflammatory reactions. It may be applied by the use of hot poultices and dressings, hot dry cloths, hot air, hot sand, irons, bricks, etc., hot-water bags, bottles, etc., hot-water coil, hot-water bath, etc. Many surgeons make a marked distinction between the use of dry heat and the employment of moist heat. The degree of warmth is also of importance, as the action of heat varies according to its degree. A moderate degree of heat, as in the case of a warm poultice, causes a dilatation of the superficial vessels, increases the hyperæmia, and thereby is of direct aid to the inflammatory reaction in increasing its protective factors, in flushing the tissue, and in increasing the exudate. A greater degree of heat may contract the

superficial arterioles and lessen the hyperæmia. It is, therefore, usually applied at the beginning of an inflammation. Its utility is doubtful and the inflammatory reaction may be delayed as the result of its application. As a rule, the use of moderate moist heat (poultices, fomentations, etc.) is often of service in the later stages of inflammation, in many cases undoubtedly hastening the process by promoting exudation and causing an earlier resolution. Such effects are seen especially in local processes that "come to a head" or "point" (suppurative inflammation). As a matter of fact, the choice between dry or moist cold and dry or moist heat probably lies wholly in the line of convenience, expediency, and comfort. Cases are, therefore, to be treated individually, so far as the employment of these agents is concerned.

Ligature of arteries supplying inflamed parts has been recommended as a means of checking inflammatory processes in certain parts of the body. Such a procedure is based upon an incorrect conception of the inflammatory reaction and must be regarded as a harmful and dangerous method of treatment. It belongs to the old antiphlogistic conception of the process ("starving of the inflammation").

Bleeding.—Venesection is now rarely employed. In certain cases, where there is a general venous stasis, its employment may be of value. Local bleeding is often resorted to when the local stasis is extreme, the part cyanotic, and the temperature lowered. This is particularly the case in severe injuries to the extremities after fractures of bones and rupture of large vessels. The stasis may be so extreme that fresh arterial blood cannot gain access to the part, and there may be serious danger of gangrene. Free incisions may relieve the venous stasis, allow the arterial blood to flow into the part, and so permit of the collateral circulation being established. The incisions should be long and deep enough to relieve the tension. Superficial scarification rarely succeeds in accomplishing this. The incisions should, therefore, be deep enough to reach through the subcutaneous tissues into the muscle. Alternating short incisions or deep punctures may be employed. The contents of hæmatomata should be removed. All of these procedures should, of course, be carried out according to the principles of aseptic surgery. In regions where it is not desirable to make incisions leeches might often be employed to advantage, but they are rarely made use of at the present time. The modern tendency is to interfere less and less with the inflammatory reaction and to devote the chief attention to the maintenance of asepsis. For that reason local incisions, like venesection, are now rarely performed.

The use of *counter-irritants*, at least in the case of acute inflammations, has also nearly become obsolete. When resolution is delayed and there is danger of the process becoming chronic, counter-irritation may often be used to great advantage. Mustard plasters, tincture of iodine, fly blisters, cutaneous irritants of various kinds, and the actual cautery may be employed. Counter-irritation

acts by the production of an active hyperæmia and the establishment of a fresher inflammatory reaction, which may aid in advancing the older sluggish process toward resolution. Exudates may in this way be more quickly absorbed, the part is flushed out by the increased circulation through it, and the processes of repair stimulated.

Compression may often be used to advantage to diminish the swelling and exudation, and thus to hasten resolution and shorten the period of convalescence. It is of particular value in the case of inflamed joints. It may be applied by means of splints, casts, elastic stockings, bandages, etc., and may be made use of both in the early and in the late stages of the inflammation. It should not be employed when the vitality of the tissues is low or when the circulation is greatly disturbed. The pressure should be applied equally and should not cause pain. The rest given to the part is probably the chief factor in the favorable results often obtained, although there can be no doubt that the absorption of exudates may be hastened by pressure. Old chronic swellings, in particular, are favorably affected by continuous pressure and often are made to disappear quickly by this means.

The local use of drugs supposed to have specific action on the inflammatory process has been practically discontinued. With the exception of bactericidal substances or of substances supposed to exert such action, local applications are now rarely made. The best surgical methods discard such treatment entirely. Aseptic methods are better than antiseptic; the agents used for bactericidal purposes may in themselves cause damage to the tissues. In the case of wounds and in operations upon parts already infected, the use of such antibacterial agents may be necessary, and in such cases those substances should be employed that cause least tissue injury or irritation. Aseptic cleansing should first be employed to the fullest extent possible, and, when thoroughly carried out, it should suffice. In the case of wounds containing foreign substances, dirt, etc., pure carbolic acid, in connection with alcohol, mercuric-chlorid solutions, etc., may be used as indicated. In the case of burns, local applications may be employed for the purpose of relieving the pain (antiseptic dusting powders, etc.). The astringent solutions so popular as local applications to inflamed parts have little or no value.

The *resolution* of acute simple inflammations is brought about by means of simple cell reproduction, healing by first intention, or by second intention, as described in detail in previous paragraphs.

2. Acute Serous Inflammations.

When the inflammatory exudate consists chiefly of fluid containing but few cellular elements, it is termed *serous* and the inflammation is spoken of as a *serous inflammation.* The collection of the fluid exudate in the tissue spaces

gives rise to an *inflammatory œdema.* Upon free surfaces the serous inflammations manifest themselves as *serous catarrhs.* Since there is usually a marked mucoid degeneration of the epithelium of such an inflamed membrane, the exudate comes to contain a large amount of mucus (*mucous catarrh.*) (See Fig. 39.) At times there is also a marked desquamation of the surface epithelium (*desquamative catarrh*). The presence of leucocytes in the exudate may give it the character of a *seropurulent* catarrh; when fibrin is present, the exudate may be classed as *serofibrinous.* Collections of serous exudate in the body cavities as the result of inflammations of the serous membranes are spoken of as *serous effusions.* Small, circumscribed collections of serous exudate beneath the horny layer of the epidermis, with the liquefaction of the lower layers of the epithe-

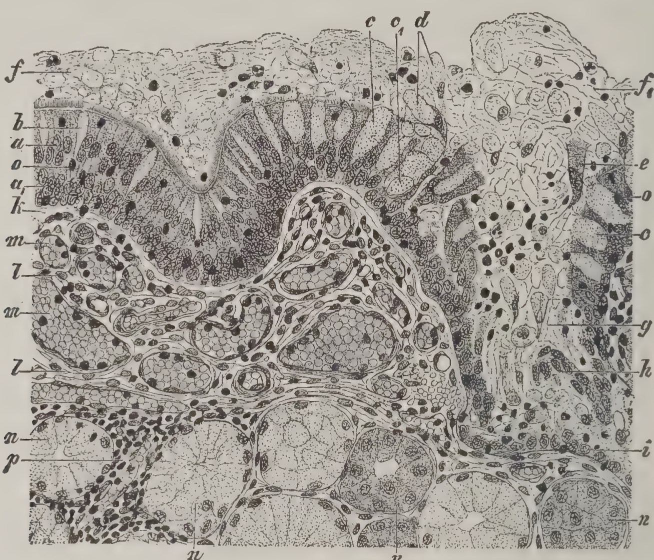

FIG. 39.—Mucous Catarrh of a Bronchus (Mueller's fluid, aniline-brown). *a,* Ciliated epithelium; a_1, deeper cell layers; *b,* goblet cells; *c,* cells showing marked mucous degeneration; c_1, mucoid cells with mucoid nuclei; *d,* desquamated mucoid cells; *e,* desquamated ciliated cells; *f,* layers of drops of mucus; f_1, layer consisting of thready mucus and pus corpuscles; *g,* duct of mucous gland filled with mucus and cells; *h,* desquamated epithelium of the excretory duct; *i,* intact epithelium of the duct; *k,* swollen hyaline basement membrane; *l,* connective tissue of the mucosa, infiltrated with cells in part; *m,* dilated blood-vessels; *n,* mucous gland filled with mucus; n_1, lobule of mucous gland without mucus; *o,* wandering cells in epithelium; *p,* cellular infiltration of the connective tissue of the mucous glands. × 110. (*After Ziegler.*)

lium, give rise to *vesicles* and *blisters.* (See Fig. 40.) Larger ones are termed *bullæ* and *blebs.*

Serous inflammations occur most frequently upon the mucous and serous membranes. They may be caused by thermal, chemical, and infective agents. The pyogenic cocci, the diplococcus of pneumonia, the colon bacillus, influenza bacillus, typhoid bacillus, bacillus of tuberculosis, etc., may produce serous, seropurulent, or serofibrinous inflammations. Inflammatory œdema may be caused by the anthrax bacillus, the colon bacillus, the gas-forming bacillus, etc. The clinical picture of a malignant œdema may result. Localized inflammatory

œdemas are also produced by certain drugs, irritant poisons, bites of insects, stings, etc. Vesicles, blebs, bullæ, and blisters may be produced by burns, corrosive poisons, chemical irritants, friction, and many forms of infection. The majority of the serous inflammations fall into the province of general medicine rather than into that of surgery. The serous catarrhs are rarely treated by the surgeon. On the other hand, the treatment of blisters, blebs, serous effusions, and inflammatory œdemas is chiefly surgical.

The *symptoms* of serous inflammations are those of inflammation in general. Since they are usually of bacterial origin, the general symptoms are more severe than in the case of a simple inflammation. The general picture of an infective process is presented, the condition running a more or less definite course. Fever and the general constitutional intoxication are more or less pronounced. The

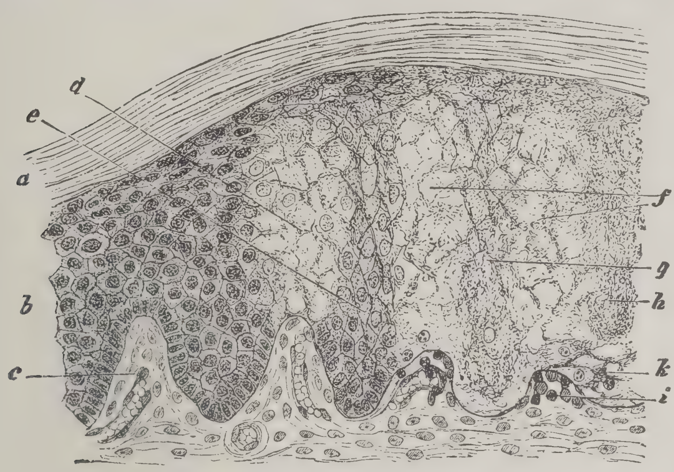

FIG. 40.—Section through the Border of a Blister Caused by a Burn (alcohol, carmine). *a*, Horny layer; *b*, rete Malpighii; *c*, normal papillæ; *d*, swollen cells, some of whose nuclei are still visible, though pale, while others have been destroyed; *e*, interpapillary epithelial cells, the deeper ones intact, those of the upper layers are drawn out longitudinally and in part are swollen and have lost their nuclei; *f*, total liquefaction of the cells; *g*, interpapillary cells, without nuclei, swollen and raised from the cutis; *h*, total degeneration of interpapillary cells which have been raised from the cutis; *k*, coagulated exudate (fibrin) lying beneath the uplifted epithelium; *i*, flattened papillæ infiltrated with cells. × 150. (*After Ziegler.*)

general principles of treatment of inflammation apply here, while treatment is also directed against the etiological agent and the extension of the process. Such general treatment usually lies outside the surgeon's field, and he is called upon to treat the purely surgical features of the case, such as the serous effusion or the blister, bleb, or bulla.

Treatment of Blisters.—Blisters or blebs when tense should be evacuated under aseptic precautions, but the epidermis should not be removed. The part may then be dressed with an antiseptic dusting powder or ointment, or dry antiseptic dressings may be applied.

Treatment of Effusions.—Serous effusions may collect in the pleural, pericardial and peritoneal cavities, the meningeal spaces, and in joints, tendon

sheaths, bursæ, etc. The amount of fluid may be so great as to cause serious pressure symptoms. The cardiac and respiratory functions may be seriously embarrassed by serous effusions in the pleural or pericardial sacs. Fatal results may be brought about by the increased pressure upon the brain or cord from the accumulation of serous exudate in the intermeningeal spaces. The movements of the joints may be seriously interfered with as the result of serous inflammations of the synovial membranes. The relief of pressure symptoms, therefore, becomes the most important indication so far as the surgical treatment of these conditions is concerned. It is a waste of time to attempt this by means of counter-irritation, attempts at specific treatment, methods of absorption, etc. Thoracentesis, lumbar puncture, aspiration of the joint cavity, etc., carried out according to the principles of aseptic surgery, yield the most certain, safe, and satisfactory results. Acute inflammations are thus kept from becoming chronic, and the secondary changes in organs from pressure are avoided. When carefully carried out, aspiration is practically without danger. In the case of large effusions, it is usually best to remove part of the fluid at one sitting and the remainder at another. The after-treatment is wholly medical, unless secondary infection should occur.

3. Acute Fibrinous Inflammations.

When the fluid exudate contains a large amount of fibrin, the inflammation is spoken of as *fibrinous*, and the exudate is classed as *fibrinous* or *serofibrinous*, as the case may be. Frequently the exudate may consist almost wholly of a thick mass of fibrin, which is deposited over the surface of the affected part (*croupous* or *membranous inflammation*). Such inflammations occur chiefly upon the mucous membranes, serous surfaces, and in the lungs, but fibrinous exudates may also be formed in tissue spaces of certain organs (lymph nodes). Fibrinous exudates occurring upon mucous membranes are often associated with a necrosis of the superficial epithelium (*diphtheritic inflammations*). The causes of fibrinous inflammations are chiefly infective agents—the pyogenic cocci, particularly the streptococcus; also the Diplococcus pneumoniæ, the Bacillus diphtheriæ, etc. The streptococcus and the diplococcus give rise chiefly to croupous inflammations of the lungs and pleura, while the bacillus of diphtheria causes diphtheritic processes in the upper respiratory tract. The streptococcus also causes diphtheritic inflammations in the respiratory tract, genito-urinary tract, and elsewhere. The irritant gases, inhalations of hot air or flame, and the corrosive poisons are also capable of producing fibrinous inflammations, chiefly of the diphtheritic type.

Upon the mucous membranes the fibrinous exudate may appear as a whitish layer or patch, or it may form a dense grayish membrane. The exudation may begin beneath the epithelium, pushing up the latter, which may degenerate or

become necrotic. The surface is then covered with a grayish membrane infiltrated with leucocytes and containing the remains of the necrotic epithelium. In other cases the exudate follows the desquamation of the fibrin. Surfaces still covered with epithelium may become covered with a fibrinous exudate from exudation occurring through neighboring denuded parts. Successive exudations may give rise to layers of fibrin pushed up from below. Crystal-like forms of fibrin may be found, usually having a leucocyte or a red blood cell as a centre. The fibrin itself is usually reticular or arranged in coarse strands lying parallel with the surface, more rarely perpendicular to it. A distinct stratification is often seen. The connective tissue below is hyperæmic, infiltrated with wandering cells, and shows an inflammatory œdema of more or less marked degree. In the tissue spaces small threads of fibrin may be found, while the dilated lymph spaces may show a thick network of fibrin threads. In the diphtheritic process the fibrin threads may be found lying between the necrotic epithelial cells. (See Fig. 41.)

Upon the serous surfaces the fibrinous exudate may appear to the naked eye as a delicate film or in the form of small granules giving the surface a rough or granular appearance, or there may be formed thick yellowish or yellowish-red deposits, which often give to the surface a felted or villous appearance (*cor villosum*). Microscopically, the fibrinous deposit may be granular or thready, or even appear in dense hyaline masses or stratified bands. The endothelium is usually desquamated in whole or in part or is necrotic. The connective tissue is more or less infiltrated with leucocytes, the blood-vessels are congested, and the tissue spaces filled with fluid containing fibrin threads. Numerous leucocytes may be present, giving the exudate a fibrinopurulent character. The villous character so often seen upon the pericardium and pleura is due to the gluing together of the opposing surfaces by the sticky fibrin and the motion of the two layers drawing the fibrin out into strings, adhesions, or into villous projections.

In the lungs fibrinous inflammations are characterized by the filling up of the alveolar spaces by a reticular network of fibrin, enclosing in its spaces leucocytes, red blood cells, and desquamated alveolar epithelium. Fibrinous exudations are also found in the kidney tubules, the bladder mucosa, endometrium, etc. In the lymph nodes and spleen fibrinous exudates may appear in the lymph sinuses of the former and the follicles of the latter.

As a rule, the fibrinous inflammations are much more severe than the serous ones, the general symptoms are more marked, and the symptoms of intoxication become of paramount importance. In the respiratory tract the disturbance of respiration becomes a feature of great importance. Diphtheritic inflammations of the upper air passages may result fatally from the stenosis caused by the formation of the membrane upon the mucous surface of the larynx, trachea, etc. Croupous inflammations of the lungs usually involve an entire lobe. There is, in consequence, embarrassment of respiration and insufficiency of the right

heart. In the case of fibrinous inflammation of the serous surfaces the symptoms are similar to those caused by serous effusions (pressure symptoms). The presence of the fibrin delays absorption, and resolution and healing are therefore retarded. The fibrin itself acts as a foreign substance or as dead material, and excites quickly the process of organization. Thickenings and adhesions consequently usually follow fibrinous inflammations. Induration of the lung

Fig. 41.—Section from an Inflamed Uvula Covered with a Stratified Fibrinous Membrane, from a case of diphtheritic croup of the pharyngeal organs (Mueller's fluid, hæmatoxylin, eosin). *a*, Surface layer of coagulum, consisting of epithelial plates and fibrin and containing numerous colonies of cocci; *b*, second layer of coagulum, consisting of fine-meshed fibrin network enclosing leucocytes; *c*, third layer of coagulum, lying upon the connective tissue, and consisting of a wide-meshed reticulum of fibrin enclosing leucocytes; *d*, connective tissue infiltrated with cells; *e*, infiltrated boundary layer of the connective tissue of the mucous membrane; *f*, heaps of red blood cells; *g*, widely dilated blood-vessels; *h*, dilated lymph-vessels filled with fluid, fibrin, and leucocytes; *i*, duct of a mucous gland distended with secretion; *k*, transverse section of a gland; *l*, fibrin reticulum in the superficial layer of connective tissue. × 45. (*After Ziegler.*)

may follow croupous pneumonia. Diphtheritic inflammations of the bladder, uterus, intestines, etc., are of importance to the surgeon, because they often follow surgical operations upon these organs. The prognosis in such cases is always grave. The streptococcus is the most common infecting agent.

The majority of fibrinous inflammations fall within the province of general

medicine rather than in that of surgery. Although often associated with or arising directly from surgical conditions, the treatment in the majority of cases is usually purely medical. Obstruction of the respiratory passages by croupous or diphtheritic membranes may necessitate surgical operations, such as tracheotomy or laryngotomy. Fibrinous inflammations of the serous membranes in the great majority of cases demand aspiration and removal of the accompanying fluid. The peritoneal cavity may be opened in the case of fibrinous peritonitis and the exudate washed out by sterile physiological salt solution. The formation of peritoneal adhesions and bands may necessitate surgical intervention because of secondary complications. In fibrinous arthritis the synovial cavity may be aspirated and the exudate withdrawn or washed out. In all cases of acute fibrinous inflammation the condition of the patient's kidneys should be carefully ascertained. Acute degenerative nephritis often brings the case to a speedy end.

4. Acute Purulent Inflammations.

When the inflammatory reaction is characterized chiefly by a leucocytic exudate, the inflammation is styled *purulent*. If the leucocyte infiltration is not so marked as to be evident macroscopically and is unaccompanied by liquefaction of the affected area, it is usually spoken of as a *small-celled infiltration*. When the leucocytes are so numerous as to give to the tissues a white, grayish, or creamy color, the infiltration is styled *purulent*. Such an exudate poured forth upon a free surface gives rise to a white or creamy, cloudy fluid called *pus*, and the inflammation is designated a *purulent catarrh*. (See Fig. 42.) A persistent and marked catarrh of this nature is often called a *blennorrhœa*. Collections of purulent exudate within the body cavities are known as *purulent effusions* or *empyemata*. A *purulent vesicle*—that is, a collection of pus beneath the horny layer of the epidermis—is known as a *pustule*. Larger collections are called *purulent blebs* and *bullæ*.

The collection of large numbers of leucocytes within the tissue spaces is usually followed by a liquefaction and dissolution of the affected area. This process is termed *suppuration*, and the resulting cavity filled with leucocytes and tissue débris is an *abscess*. The contents of the cavity are also designated as *pus*. When the suppurative process occurs upon the surface of the skin or mucous membranes, there is a superficial loss of substance giving rise to an *ulcer*. The process of suppuration, when extending through the tissues, often gives rise to duct-like tracts known as *fistulas* or *sinuses*.

The inflammatory exudate often assumes the character of a *seropurulent* inflammation when the fluid portion is abundant. The infiltration of the tissues by such an exudate gives rise to a *purulent œdema*. (See Fig. 43.) Purulent and seropurulent inflammations, when rapidly involving large areas, particularly of the subcutaneous or subserous tissues, are designated as *phlegmons* or *phlegmo-*

nous inflammations. Large pus cavities may thus be formed. The presence of fibrin in purulent or seropurulent exudates gives rise to a *fibrinopurulent inflammation.* Such inflammatory exudates are of very common occurrence in the serous cavities, lungs, and upon mucous membranes. The exudate of an inflammatory œdema or phlegmonous inflammation often bears this character.

Suppuration.—The steps of a suppuration may be traced as follows: There is, first, a primary tissue lesion, either degenerative or necrotic. This is followed by hyperæmia, marginal disposition of the leucocytes, collection in the tissues of mononuclear cells, diapedesis of leucocytes with polymorphous nuclei, phagocytosis, and increasing emigration of the leucocytes until the tissue be-

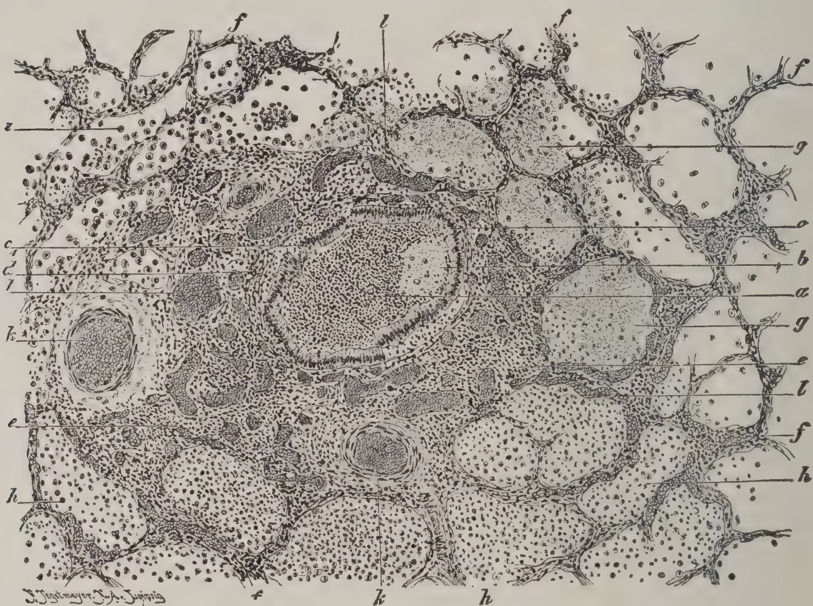

FIG. 42.—Purulent Bronchitis, Peribronchitis, and Peribronchial Bronchopneumonia in a Child one year and three months old (Mueller's fluid, hæmatoxylin-eosin). *a*, Purulent; *b*, mucoid bronchial contents; *c*, c_1, bronchial epithelium infiltrated with round cells and partly desquamated; *d*, infiltrated bronchial wall with greatly dilated blood-vessels; *e*, infiltrated peribronchial and periarterial connective tissue; *f*, alveolar septa, in part infiltrated with cells; *g*, fibrinous exudate in the alveoli; *h*, alveoli filled with exudate rich in cells; *i*, alveoli filled with exudate containing few cells; *k*, cross-section of a pulmonary artery; *l*, bronchial, peribronchial, and interacinous vessels showing marked congestion. × 43. (*After Ziegler.*)

comes densely packed. The injured or necrotic tissue elements now undergo a liquefaction, while the leucocytes contained in the fluid thus formed begin to degenerate. Both primary and secondary tissue damage, therefore, usually occurs in an area of suppuration. The tissue may be killed at once by the injurious agent or it may be damaged, to die later during the process. The leucocyte collection, the fluid exudate, the abnormal conditions of pressure and nutrition, the disturbance of relationship, possibly also chemical substances produced by the body cells, etc., play a part in the secondary liquefaction which

always distinguishes a suppurative process. It is very probable that the leucocytes play a chief part in this dissolution of the dead or damaged tissue. In all acute suppurative processes and in all exudates rich in leucocytes, peptones and albumoses are found, and their formation may be the result of an extracellular action of the leucocytes. Such a "digestion" of the dead area may also be interpreted as protective or reparative. Upon a surface the dead tissue is usually cast off before complete liquefaction has taken place. In demarcating inflammations the process of liquefaction takes place only at the periphery of the dead area where the leucocytes have assembled. The involvement of the leucocytes in the liquefaction process may be explained by the abnormal conditions under which the cells are placed, the failure of reproduction, bacterial substances producing leucolysis, etc. When the area of necrosis is very large the process of suppuration is usually incomplete and takes place only at the periph-

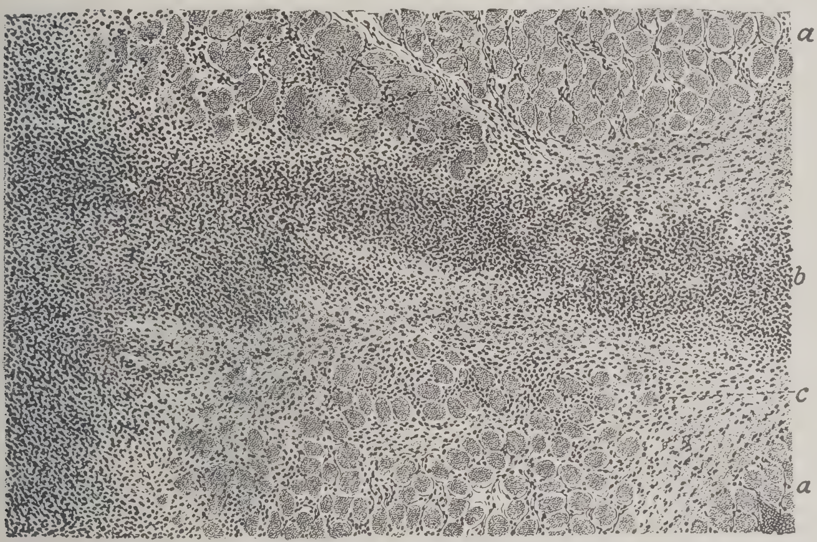

Fig. 43.—Hæmatogenous Staphylococcus Myositis (alcohol, hæmatoxylin-eosin). *a*, Transversely cut muscle bundles; *b*, purulent; *c*, seropurulent, partly coagulated exudate. × 45. (*After Ziegler.*)

ery of the necrotic tissues. In the case of deep-seated areas of this kind, there is left behind a fatty débris which ultimately undergoes caseation or liquefaction, or may become inspissated and impregnated with lime salts. Histolysis—that is, tissue liquefaction—is the essential feature of suppuration, and this is brought about by proteolytic ferments produced by the body cells and the bacteria.

Pus.—The purulent exudate upon a free surface and the product of suppuration are both called pus. It appears, ordinarily, as a creamy fluid, more or less mucoid, having usually an alkaline reaction, although not infrequently acid, and having a peculiar sweetish odor. When poured into a glass cylinder, pus commonly separates into two layers—the upper one consisting of a transparent, yel-

lowish fluid ("*liquor puris*"), while the lower layer is thick, opaque, whitish or yellowish in color, and consists of the more solid constituents. The upper layer resembles the lymph and blood serum in its composition. The albumin content is generally somewhat lower, but it may be higher. Fibrinogen, as a rule, is not present, so that pus ordinarily does not coagulate. Globulin, albumose, leucin, tyrosin, and other extractives, more or less mucin or pseudomucin, fats, cholesterin, etc., are found in pus. The chief salts present are sodium chloride and magnesium and calcium phosphate. Proteolytic ferments, antibacterial and antitoxic substances, arising from the bacteria and from the body cells, are also contained in the serum of pus. The specific gravity varies from 1.030 to 1.033.

As might be expected from the varied etiology and the varying conditions under which pus is formed, both its macroscopical appearances and its chemical composition vary greatly. It may be thin ("*ichor*"), having a low specific gravity and containing flakes and shreds of fibrin. Lactic, butyric, valerianic, and other organic acids may be contained in it and give it their characteristic odor. Hydrogen sulphide (H_2S) may be present in it, and pus containing so many gas bubles as to give it a foamy appearance may be seen in case of infections with the gas-forming organisms. A very foul odor may be occasioned by the growth of putrefactive organisms. The presence of blood may give it a bright red or brown or chocolate color (*sanies*). A blue or green color may be given to pus by the Bacillus pyocyaneus. An orange-colored pus may be produced by a deposit of crystals of hæmatoidin. Red pus occurs rarely as the result of the presence of a large chromogenic bacillus. It may be distinguished from bloody pus by the fact that the red color does not change upon the dressings when dry, while blood soon takes on a brown color. A fecal color and odor may be present in pus in the peritoneal cavity, or such pus may be bile-stained.

The chief cellular constituent of pus is the polynuclear leucocyte. A pus cell is nothing more than a leucocyte. In fresh pus the nuclei of the pus cells may stain as well as those of the cells of the inflammatory infiltration. Usually after suppuration is established numerous degenerating cells are found in the pus, their protoplasm showing fatty and granular degeneration. In old pus nearly all of the pus cells may show karyorrhexis or karyolysis. Besides the polymorphonuclear leucocytes there may also be found in pus eosinophile cells, large hyaline mononuclear cells, as well as cells of the small lymphocyte type. Occasionally the mononuclear cells may predominate. The older the process the greater the proportion of mononuclear cells, as a rule. The nuclei of the pus cells are usually very irregular in shape, probably as the result of amœboid motion at the time of fixation, or the varied nuclear shapes may be due to beginning karyorrhexis. Round, oval, or spindle cells, arising from the proliferation of the fixed connective-tissue cells or endothelium, may also be present in the pus. There is as yet no method of distinguishing between the round cells of the lymphocyte type and those arising from the tissue cells. Cellular detritus

resulting from the primary tissue lesion and the suppurative process are also contained in pus. Shreds of tissue, blood, blood pigment, fibrin, parasites, foreign bodies, caseous and calcareous masses, cyst contents, hyaline bodies, etc., may at times be found in pus. The cells of the pus produced by pyogenic organisms are of the same character as those occurring in pus produced by means of chemical irritants. In so far as the body reaction is concerned, there is absolutely no difference in the morphology of the two kinds of pus formation.

Since purulent reactions are in the great majority of cases due to pyogenic infections, there may be found in the pus, as a rule, the organisms producing it. Their presence may be demonstrated by cultural methods or by stained preparations of the pus. In the case of some organisms—actinomyces, for example —grayish or yellowish granules are formed by the organism, and these may be seen macroscopically. In many cases, however, the pus is sterile, the infecting organisms having been wholly destroyed. The death of the bacteria causing the purulent reaction is ascribed to the action of bactericidal substances produced by the bacteria themselves or by the body cells. The latter source is the more important. Sterile pus possesses bactericidal properties to a greater extent than does normal blood serum or lymph. We must believe that pus, in its essential elements, leucocytes and serum, is protective. Phagocytosis and the formation of antibodies constitute its chief functions. In this sense, then, all pus is *laudable.* Inasmuch as the purulent reaction varies in degree according to the virulence of the infective agent, pus has come to be itself regarded as the harmful agent. The etiological agent should not be confounded with the reaction to the injury produced by it. Pus may be dangerous, in that it may contain the pyogenic organisms or because of certain conditions favoring secondary tissue damage; but the essential biological fact should not be lost sight of—*the production of pus is a protective reaction to injury.* The limitation of the term pus to that pus alone which contains pyogenic bacteria is a purely arbitrary usage and not practical. Hueter's dictum, that pus can be produced only by pyogenic organisms, has been many times disproved. Further, in a great many cases of infection with pathogenic bacteria, the latter, by the time suppuration has occurred and pus has formed, have been entirely destroyed, and the resulting pus is sterile. Further, the so-called pyogenic organisms may give rise to simple, serous, or fibrinous reactions, instead of purulent. Inasmuch, however, as clinically purulent inflammations are almost without exception due to micro-organisms, it is easily understood why many clinicians come to regard the *process* and the *infective agent* as having the same significance.

The purulent reaction is due, in the great majority of cases, to infection with the *Staphylococcus aureus, albus,* and *citreus* and the *Streptococcus pyogenes.* Next to these, rank as the most common pyogenic organisms the *Diplococcus pneumoniæ, Bacillus mucosus capsulatus, Bacillus coli communis, Bacillus pyocyaneus, Bacillus typhi abdominalis, Bacillus influenzæ,* and *Actinomyces.* Other

organisms more rarely exciting purulent reactions are *Micrococcus tetragenus, bacillus of chicken cholera, bacillus of swine plague, Micrococcus intracellularis, Bacillus prodigiosus, Proteus Zenkeri, Micrococcus pyogenes fœtidus, Bacillus mallei*, a variety of *Blastomycetes* not yet classified, *Oidium albicans, Tricophyton tonsurans, Sporothrix Schenckii, Bacillus aërogenes capsulatus, Bacillus anthracis, Bacillus tuberculosis*, etc. Of the animal parasites, the *amœba* of dysentery is associated in such a way with abscess of the liver as to make it very probable that it is the etiological factor.

Among the *chemical* substances that produce a purulent reaction when introduced into the tissues are mercury, oil of turpentine, creolin, croton oil, silver nitrate, petroleum, zinc chloride, digitoxin, bacterial proteins, also animal and vegetable proteins. Practically, such chemical suppurations are almost wholly experimental and are rarely met with clinically except as the result of hypodermic injections. The suppurations produced by chemical agents are histologically and biologically exactly the same as those produced by pyogenic organisms. They differ from the latter only in that they do not contain infective agents capable of indefinite growth, that they heal more easily, do not spread, and do not give rise to metastasis.

The common pyogenic organisms are constantly present upon the skin, in the respiratory and genital tracts. A lowering of the local resistance, as through a wound, is usually necessary for infection. The occurrence of suppuration is, however, favored by acute and chronic infectious diseases, chronic valvular diseases, diabetes mellitus, etc. Variola, scarlatina, diphtheria, typhoid fever, gonorrhœa, measles, dysentery, and influenza predispose greatly to secondary infections with the *Streptococcus pyogenes* and other pyogenetic bacteria. Mixed pyogenetic infections are not uncommon, the staphylococcus and the streptococcus being most frequently associated.

The general symptoms of purulent inflammation are more marked than those of simple reactions. The affected area is greatly swollen, tense, and brawny, and of a bright red color. The local pain is severe and of a throbbing or boring character. In the case of suppurative processes there is usually a chill or there are repeated chilly sensations, with a sudden rise of temperature. The fever usually persists until the pus is discharged. In the case of an abscess on the surface of the body, the advent of suppuration is shown by the softening of the centre of the inflamed area and by fluctuation. At the centre of the soft area a light-colored spot appears, which ultimately ruptures by "pointing." After the free discharge of pus the general and local symptoms gradually diminish. The hyperæmia disappears, and the swelling lessens so that the skin becomes wrinkled, and the pain ceases. Should the fever and other constitutional symptoms persist, an extension of the process locally may be taking place or metastasis of the infective agent has occurred.

Purulent inflammations show a tendency to spread in the direction of least

resistance. They may be subfascial or subperiosteal and spread along beneath the fascia or the periosteum. In the muscles the inflammation extends along the intermuscular connective tissue. It also follows along the blood-vessels and nerve trunks. The symptoms of deep suppuration develop more gradually than when the process is superficial. Pain and fever are the first signs; there may be no swelling or surface redness. The surface of the inflamed region may then become œdematous, and later red and tender. As the process approaches the surface the symptoms become more marked and characteristic.

The condition of pyæmia or septicæmia may develop in the case of any local purulent inflammation. The constitutional symptoms become correspondingly more severe, the fever more marked, chills more frequent, and a typhoid state may supervene. The clinical picture may be further marked by the effects of the toxins upon the heart muscle, kidneys, etc. The case may finally terminate in cardiac insufficiency or uræmia. The purulent process may spread diffusely throughout the tissues (phlegmon). When it occurs on a body surface, there is seen an advancing line of redness and swelling. The constitutional symptoms are usually marked. The involvement of the regional lymphatics is, as a rule, a dangerous matter and demands prompt and energetic treatment. The primary infection is often insignificant or the entrance of the infective agent is not noticed. The superficial lymphatics running from the point of entrance become swollen and palpable, and appear as red lines or cords. The lymph nodes are swollen and tender.

Treatment of Purulent Inflammations.—The chief indication is the removal or destruction of the pyogenic agent, and the prevention of its spread by extension or metastasis. Prompt intervention by the surgeon is demanded. The developing colony of bacteria must be reached where possible, and vigorous antiseptic measures carried out against it. Free incisions, scraping, curetting, excision, antiseptic douches, etc., are among the methods that may be carried out to this end. Antiseptic poultices and baths are also of value. In the case of extensive involvement it is often better to make multiple incisions in such a way as to secure satisfactory drainage. The incisions should be made where the resulting scar will not cause disfiguration or interfere with the function of the part.

The strength of the patient must be kept up by nutritious and easily digestible food. Alcohol may be given. Strychnine and digitalis should be given according to the state of the heart's action. The patient should be kept in bed. The local symptoms may be met according to the methods mentioned above for the treatment of simple inflammations.

Purulent Catarrhs.—The majority of these conditions are treated by the physician rather than by the surgeon. The chief form of purulent catarrh usually coming within the field of surgical practice is that of gonorrhœal infection. Purulent catarrhs of the bladder and upper urinary tract are also often treated

by the surgeon. In general, the local treatment of these conditions consists of antiseptic or aseptic irrigation, injections, etc., while the general indications are met according to general principles.

Abscesses.—The term abscess is applied to the results of suppuration within the body tissues; that is, to a cavity filled with the products of the liquefaction of an inflamed area (pus) (see Fig. 44). It is one of the most common forms of purulent inflammation. There are numerous clinical varieties, and many designations are applied to them according to their location, character, duration, etc. The term abscess is also applied by many writers to the collection of pus in the body cavities. Others prefer to class these as *purulent effusions* or *empyemata*, or simply to use the terms indicating the region involved (purulent pericarditis,

FIG. 44.—Embolic Abscess of the Intestinal Wall with Embolic Purulent Arteritis, and Embolic Aneurism in Cross-section (alcohol, fuchsin). *a, b, c, d, e,* Layers of intestinal wall; *f*, remains of arterial wall, cross-section; *g*, embolus, surrounded by pus corpuscles lying within the dilated and partly suppurating artery; *h*, parietal thrombus; *i*, periarterial purulent infiltration of the submucosa; *k*, vein showing marked congestion. × 28. (*After Ziegler.*)

peritonitis, etc.). Pelvic abscess is used to indicate a collection of pus in the pelvis shut off by adhesions. Subdiaphragmatic abscess is applied usually to a localized purulent peritonitis with a collection of purulent fluid between the diaphragm and neighboring organs, usually the liver. Special names, such as *boil, furuncle, carbuncle, whitlow, felon*, etc., are applied to certain forms of abscess.

Abscesses may vary in size from those which are microscopic or "pin-point" to those containing one or two litres of pus. Abscesses containing from four to six litres have been reported. The large abscesses are usually found in the subcutaneous or subserous tissues or in the intermuscular fascia. The wall of an

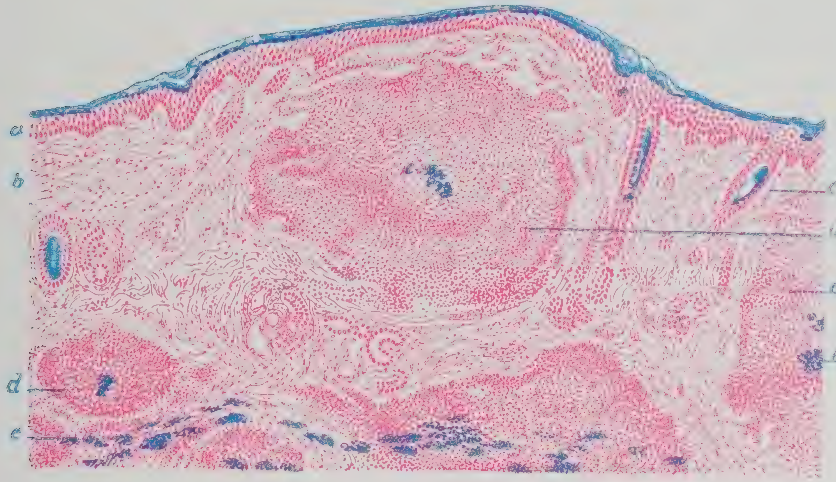

FIG. 1

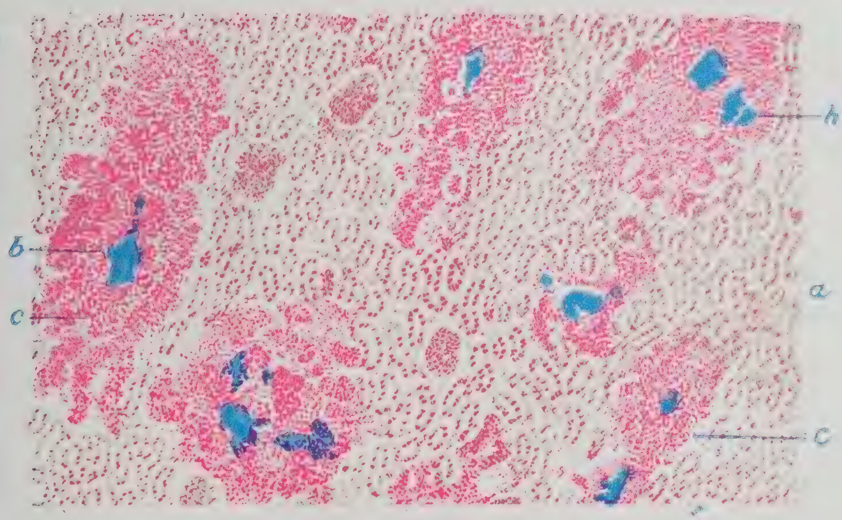

FIG. 2

ACUTE PURULENT INFLAMMATION; ABSCESS.

(*After Ziegler.*)

FIG. 1.—Multiple Abscesses of the Skin, due to Staphylococci. (Alcohol, carmine, Gram's method.) Child of three weeks. *a*, Epithelium ; *b*, corium ; *c*, hair-follicle ; *d*, *e*, purulent foci with cocci. × 40.

FIG. 2.—Miliary Purulent Nephritis. Caused by Staphylococci, primary focus in skin (furunculosis. (Alcohol, methyl-violet, carmine.) *a*, Normal kidney tissue ; *b*, collections of cocci ; *c*, purulent focus. × 43.

acute abscess is made up of more or less degenerated tissue elements infiltrated with pus cells. The abscess may be sharply circumscribed or the suppurative process may extend ("burrow") along the paths of least resistance. As a rule, all abscesses tend toward a surface, where they "*point*" or "come to a head." In the case of abscesses of the internal organs, the rupture may occur into any one of the hollow organs or body cavities or passages. Adhesive inflammatory reaction about the burrowing pus may prevent such rupture. Abscesses that do not rupture spontaneously or are not incised may, after the death of the pyogenic organism, become organized, calcified, or converted into a cyst. Large abscesses become encapsulated. Healing of an abscess takes place through the proliferation of the cells of the abscess walls and the formation of a granulation tissue which gradually fills up the cavity. The processes of repair are aided by the evacuation of the pus and dead material and the apposition of the abscess walls.

Metastatic or *embolic* abscesses arise from the transportation through the blood or lymph of the infective agent. (See Fig. 44.) Since they are often small, they are frequently called "pin-point" or "pin-head" or "miliary abscesses." (See Plate I., Fig. 2.) The occurrence of secondary foci of infection and suppuration constitutes the condition of *pyœmia.* It occurs most frequently in the case of infections with the staphylococcus, streptococcus, Micrococcus lanceolatus, Bacillus mucosus capsulatus, and Actinomyces.

The treatment of abscesses is to be discussed farther on, in a separate article devoted to this subject.

Among the most common forms of abscesses are those which occur in the skin and subcutaneous tissues, known as *pustules, boils, carbuncles, felons,* etc. (See Plate I., Fig. 1.) *Pustules* occur most commonly as the acne pustule, and are the result of infection of the hair follicle or sweat gland, with resulting obstruction of the duct. The *boil* or *furuncle* differs from the pustule only in the virulence of the infection and the depth to which the inflammation extends. The bacteria gain entrance through the hair follicles or the sweat glands. Through the growth of the infecting organisms and their formation of toxins there results an area of coagulation necrosis, which forms the "core of the boil." The part usually thus destroyed is the hair follicle and its sebaceous gland. The first symptom of the boil is the formation of a small pustule in a hair follicle, accompanied by an itching sensation. There quickly results more or less infiltration of the neighboring skin and subcutaneous tissues, and the boil becomes very sore and tender on pressure. A crust then forms at the site of the pustule. This, when removed, usually shows a well-defined circular opening from which pus exudes. Into the opening a probe may be passed for some distance. The suppuration increases, and after a few days the core is expelled and the cavity heals by the formation of granulation tissue. The staphylococcus is the most common etiological factor, although streptococcus

boils are not rare. Boils not infrequently appear in succession (*furunculosis*). The patient usually infects himself through scratching, but in these cases there is generally some lowered resistance of the body tissues.

A *carbuncle* is an infective, suppurative, and gangrenous inflammation of the skin and subcutaneous tissues, beginning as a boil and spreading gradually downward and laterally in the subcutaneous tissue. It differs from a boil only by the extent of the tissues involved and by the multiple points of suppuration. Staphylococci are the most common bacteria found in carbuncles. They occur usually in adults and old persons. As a rule, they are situated upon the back of the neck, although occasionally found elsewhere. A fully developed carbuncle has a broad, flat base. Over it the skin is elevated, reddened, and ex-

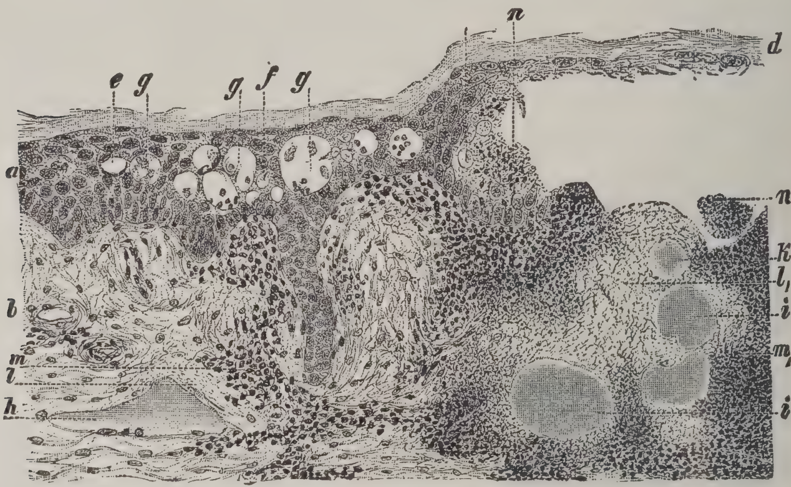

Fig. 45.—Section of the Skin in Erysipelas Bullosum (alcohol, alum-carmine). *a*, Epidermis; *b*, corium; *c*, vesicle; *d*, covering of vesicle; *e*, epithelial cells containing vacuoles; *f*, swollen cells with swollen nuclei; *g*, g_1, cavity caused by the liquefaction of epithelial cells, and containing fragments of epithelium and pus corpuscles; *h*, lymph-vessel, partly filled with streptococci; *i*, lymph-vessel filled with streptococci; *k*, colony of streptococci in the tissue; *l*, *l*, necrotic tissue; *m*, cellular, m_1, fibrinocellular infiltration; *n*, fibrinocellular exudate in the vesicle. × 60. (*After Ziegler.*)

tremely tense. Through the skin there may develop a number of openings from which pus oozes. These may become confluent into one or more larger openings through which large subcutaneous sloughs may be seen. Carbuncles are often several inches in diameter. They reach their full development about the end of the second week, but the process of healing may be delayed over a number of weeks. The larger carbuncles give rise often to very grave constitutional disturbances and not rarely result fatally. When occurring in association with diabetes the prognosis is especially grave.

Panaritium or *felon* is a variety of subfascial abscess occurring in the fingers or hands. According to its location it may be classed as cutaneous, tendinous, subperiosteal, or palmar. The infection occurs through some slight skin injury, such as an abrasion, blister, callus, punctured wound, cut, etc.

Cooks, dish-washers, dissectors, etc., are especially liable to these forms of infection. The felon occurs most frequently at the ends of the finger. The symptoms are intense, throbbing pain, with a gradually increasing swelling, more or less fever, and symptoms of general intoxication. The complications, sequelæ, and the prognosis of felons depend upon the relations of the felon to the structures of the part involved. Tendinous felons may destroy a phalanx or seriously injure a joint. More serious results of the same nature are caused by the subperiosteal felon. Lymphangitis and secondary involvement of the regional lymph nodes are especially likely to occur in association with felons.

Phlegmonous inflammations are those characterized by rapid and diffuse spreading through the tissues (see Figs. 45 and 46). They are usually the result of a streptococcus infection. The signs of an acute inflammation are present over a large area. Sloughing takes place early and suppuration is soon established. Felons not infrequently give rise to such processes. The whole arm may be quickly involved, the skin becoming hard and brawny, covered with blebs, and the tissues of the limb as a whole very œdematous. Occasionally the process resembles that of a malignant œdema, the subcutaneous tissue becoming emphysematous. This condition is probably the result of an infection with the *Bacillus aërogenes capsulatus.* The occurrence of a true malignant œdema in man is still unsettled. The constitutional symptoms of phlegmonous inflammations are usually marked. The condition of *septicæmia* is ordinarily present at the same time. The treatment is the same as that for abscess.

Ulcer.—The term ulcer has been rather loosely applied by different writers to a number of conditions which resemble each other in that there is a loss of continuity of a surface, either that of the skin or that of a mucous membrane. The results of suppuration, superficial necrosis, granulating wounds, etc., have all been included under this head. The pathological picture ultimately presented by these conditions is the same. In a broad sense, then, ulcers might be defined simply as a loss of continuity or a superficial loss of substance of the skin or mucous membrane, due to some form of tissue lesion. Clinically, however, the term has come to convey the impression that the loss of substance is the result, either wholly or in part, of the inflammatory reaction—that is, the result of suppuration or a demarcating inflammation. The term ulceration is used by some writers as a synonym for ulcer, by others to indicate an extensive process or the occurrence of multiple ulcers, while in the pathological usage of the term it indicates the process rather by which the ulcer is formed.

The clinical variety of ulcers is very great. They are classed according to the etiology, their location, and their characteristics of spreading, healing, etc. According to etiology they are usually classed as *non-specific* ulcers, *specific*, and *malignant.* The non-specific ulcers include all those cases which are not due to some specific infection or to malignant disease. They are the result of trauma, infection with pyogenic or saprophytic bacteria, anæmia, pressure,

local or constitutional disease, etc. The specific ulcers are those occurring in syphilis, tuberculosis, dysentery, typhoid fever, diphtheria, glanders, malaria, actinomycosis, blastomycosis, leprosy, etc. The malignant ulcers arise through the degeneration or infection of a superficial malignant tumor. Carcinoma of the mouth cavity, œsophagus, stomach, intestine, and uterus, epithelioma, and rodent ulcer are the most frequent malignant tumors giving rise to ulcer. Sarcoma of superficial parts less frequently ulcerates. A malignant growth may also arise in a chronic ulcer, the so-called "malignant degeneration" of an ulcer.

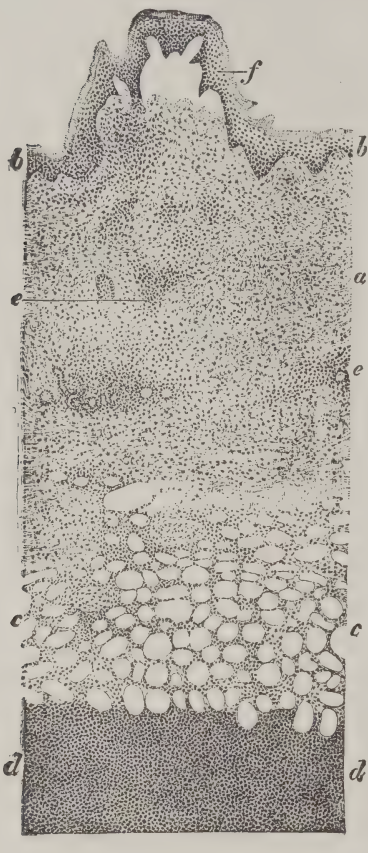

FIG. 46.—Phlegmon of the Subcutaneous Tissue, with Formation of a Vesicle through Œdema (Mueller's fluid, hæmatoxylin, eosin). *a*, Corium; *b*, epidermis; *c*, infiltrated fat tissue; *d*, focus of pus; *e*, cellular foci in corium; *f*, subepithelial vesicle due to œdema. × 30. (*After Ziegler.*)

According to their course ulcers are classed as *acute, subacute,* and *chronic.* According to their condition they are described as *healing, spreading, inflamed, phagedenic, sloughing, serpiginous, indolent, fungating, scirrhous, hemorrhagic,* etc.

Ulcers occur more often in adult life and in old age. They are much more frequent in men than in women, and are generally seen in individuals of the lower classes. These facts are easily explained by the importance of trauma, syphilis, and the occurrence of infection as the etiological factors. Many constitutional diseases, such as diabetes, scurvy, syphilis, tuberculosis, anæmia, etc., predispose to the occurrence of ulcers. The acute infections, particularly typhoid fever, variola, and scarlatina, favor the development of ulcers from exciting causes that otherwise usually are without effect. Chronic diseases, such as chronic valvular lesions of the heart, fatty heart, arteriosclerosis, obesity, etc., similarly predispose to the formation of ulcers. Local predisposing causes may be found in any thing interfering with the arterial circulation, the venous or the lymphatic circulation. Vasomotor disturbances often play an important part in the development of ulcers. Certain forms of skin diseases (herpes, ecthyma, pemphigus, eczema, etc.) are frequently associated with ulceration. Ulcers may also be produced by the elimination of certain drugs (ulcerative stomatitis and ulcerative colitis caused by mercurial poisoning, etc.).

The bacteriology of ulcers is extremely varied. Nearly all the pathogenic micro-organisms may be found as etiological agents in the production of ulcera-

tion. The typhoid bacillus, Shiga bacillus, diphtheria bacillus, the amœba coli, the malarial plasmodium, etc., are among the specific agents which may under certain conditions give rise to ulcers. Not infrequently the only micro-organisms found in ulcers are saprophytic bacteria.

Microscopically, an acute ulcer shows a superficial loss of substance with an infiltrated base and edges. Over the base there is a layer of exudate and tissue débris. Sooner or later proliferation of the connective-tissue cells at the periphery gives rise to the formation of granulation tissue, and the ulcer heals by second intention. The prolongation of the healing process gives rise to the formation of scar tissue about the ulcer and a hyperplastic condition of the bordering epithelium.

The most common forms of ulcer seen by the surgeon are the *syphilitic*, *varicose*, *traumatic*, and *pressure ulcers* (*decubitus*). The treatment of ulcers is both general and local. The constitutional treatment consists in the support and building up of the body by means of proper diet and hygiene, tonics, etc. The local treatment is aimed at the cleansing and sterilization of the ulcer, the stimulation of repair, and the control of the reparative process. In the case of a recently formed ulcer the local treatment is that carried out in any suppurative process, being directed against the infective agent and also aimed at the relief of the inflammatory symptoms. For further details the reader is referred to the article on Ulcers and Ulceration.

Fistula, Fistulous Tract, and Sinus.—An abnormal opening into a normal body cavity or organ is known as a *fistula*. The term is also applied to congenital openings or defects as well as to openings produced by suppurative processes. When, as the result of a "burrowing" suppuration, there is formed a long, narrow channel, the latter is designated a *fistulous tract* or *sinus*. These conditions are usually characterized by a failure of the healing process. They are often due to the presence of a foreign body, infected ligature, etc., or the position of the fistula is such that the body movements keep it from healing. Physiological secretion (urine, saliva, fæces, bile, etc.) may serve to keep the fistula from closing. Further, many fistulas are the result of tuberculous infection.

When the fistulous tract or sinus is superficial, it should be opened by a free incision and its surfaces curetted. Foreign bodies, ligatures, etc., should be carefully sought and removed. Small tuberculous fistulas may be removed entire. In other cases a careful dissection of the wall may be carried out. Treatment with antiseptic washes, irrigation, injection, etc., may be instituted according to indications. As the fistula and sinus show little disposition to heal and so tend to run a protracted course, careful search should be made to ascertain the cause and source of the condition, and all diseased tissue should be thoroughly removed. Constitutional treatment is often of the greatest importance, particularly when tuberculous infection is suspected.

5. Acute Degenerative and Necrotic Inflammations.

When an organ or tissue presents extensive parenchymatous degeneration, such as *cloudy swelling, fatty degeneration,* etc., without evidences of an inflammatory reaction, the condition is usually spoken of, both clinically and pathologically, as that of "*acute degenerative parenchymatous inflammation.*" In reality it represents the tissue lesion alone, without any inflammatory reaction as yet having been initiated. Such processes are seen particularly in the liver and kidneys, and are usually the result of intoxications, either from bacterial infections or from chemical poisons. The use of the term inflammation is, of course, not justified from the pathological standpoint, and it would be more proper to class them as degenerations. The fact that no inflammatory reaction is found is, however, due to the death of the patient before it has had time to develop; in fact, all stages may be found in different cases, from the pure degeneration or necrosis to a fully developed condition of inflammation. There is, therefore, a certain practical reason for classing all these conditions under the head of degenerative inflammation. To the surgeon these conditions are of great importance, since they most frequently arise from the absorption of bacterial toxins from some local focus of infection. To an acute degenerative nephritis or myocarditis the fatal termination in septicæmia, septicopyæmia, and sapræmia is usually due. The treatment is chiefly preventive. The control of the local infection, its restriction from spreading, the promotion of excretion of poisons absorbed, etc., are the chief indications of treatment.

Some of the injurious agents acting upon the body produce a tissue lesion of the nature of extensive necrosis. The necrotic tissue remains unchanged for a long time, and is only rather late removed by means of sequestration, sloughing, absorption, etc. In such cases the tissue necrosis, therefore, becomes the most striking feature of the process, and such inflammations are known as necrotic inflammations. The necrosis may be apparent before the inflammatory reaction, as in the case of burns, corrosive poisons, freezing, anæmia, etc. In other cases, particularly in infections, the inflammatory reaction may first be seen, the inflamed and infiltrated tissues later becoming necrotic. Tuberculosis may be taken as an example of a necrotic inflammation of the latter type; as a rule, the caseation necrosis occurs after tissue proliferation has existed for some time. Necrotic inflammations are caused chiefly by high or low temperatures, anæmia, caustics, and infection. In the case of the action of high or low temperature and anæmia, the tissue necrosis occurs in the part involved. Corrosive poisons also act locally, but many poisons produce necrosis not only at the point of contact, but in other portions of the body as well, after their diffusion through the blood or lymph. Mercury, cantharidin, the salts of chromic acid, etc., cause necrosis in the intestines, urinary passages, and kidneys, as well as at the points with which they first come in contact. Bacteria cause necrosis both at the place

where they multiply and in those portions of the body where they are excreted after being absorbed.

Necrotic inflammations are most frequently seen on the mucous membranes, and are usually called *diphtheritic inflammations* or *diphtheritis* (see Fig. 47). As a rule, the latter designation is applied only to those processes in which the infiltrated subepithelial connective tissue is also involved in the necrosis, but necrosis of the epithelium alone is often spoken of as *epithelial* or *superficial diphtheritis*. The necrotic epithelium may be recognized by the occurrence of white opaque patches. In a true diphtheritis the entire epithelial surface is necrotic as well as the upper layers of connective tissue, the dead parts becoming changed into a lumpy or granular mass without nuclei, or into a hyaline mass containing fibrin. Usually no evidences of structure can be made out in the dead mass. This constitutes the so-called *diphtheritic membrane*. Such processes occur most frequently in the intestines (dysentery, diphtheritic colitis, etc.), in the vagina and uterus (diphtheritic vaginitis and endometritis), in the descending urinary tract and bladder (diphtheritic ureteritis and cystitis), and in the upper respiratory tract (diphtheria of tonsils, fauces, larynx, etc.). In the respiratory tract the process is usually caused by the *Bacillus diphtheriæ*, in the intestinal and genito-urinary tract by the *Streptococcus*. The other pyogenic organisms are capable under certain circumstances of producing a similar necrosis. An infection of a wound by virulent bacteria (*Staphylococcus* or *Streptococcus*, etc.) may produce a similar necrosis of the wound granulations (*wound diphtheritis*). Necrotic inflammations may also occur within the internal organs as the result of infection. The lymph nodes, spleen, and bone marrow are most frequently involved.

FIG. 47.—Bacillary Diphtheritis of the Large Intestine in Dysentery (alcohol, gentian-violet). *a*, Necrotic portion of the glandular layer of the mucosa, infiltrated with bacilli; *b*, intact inflamed mucosa; *c*, muscularis mucosæ; *d*, submucosa; *e*, colonies of bacilli; *f*, glands with living epithelium; *g*, glands with necrotic epithelium and bacilli; *h*, connective tissue infiltrated with cells; *i*, blood-vessels. × 80. (*After Ziegler.*)

The diphtheritic inflammations of most interest to the surgeon are those occurring after operations upon the intestinal and genito-urinary tracts and upon wounds. The streptococcus is the most common etiological agent. The process is very severe, the infection virulent, and the cases run usually an unfavorable

course. Diphtheritic colitis and cystitis are very frequently the immediate cause of death. The latter usually occurs after operations for stone, stricture, and enlarged prostate. The treatment is chiefly preventive. After infection of this nature has occurred and the process has become established, the treatment is in general the same as that for suppurative inflammations.

If an area of inflammation becomes infected with bacteria capable of producing putrefaction, the inflammatory process assumes the character of a putrid gangrene and the inflammation is designated as a *gangrenous inflammation.* The term *gangrene* alone is usually applied to the condition. While the pathologist uses this term to denote a necrosis accompanied by *putrefactive* processes, the surgeon often uses it to designate simply the death of tissues *en masse*—that is, to signify an extensive necrosis of an exposed portion of the body. As a matter of fact, such a superficial death of tissue is practically always accompanied by decomposition, so that no essential contradiction exists in the different applications of the term. The presence of saprophytic or putrefactive bacteria may, therefore, be regarded as the most distinctive feature of gangrene.

Gangrenous inflammations may be either *primary* or *secondary*. The *primary* form is due to infection with some specific micro-organism, and is to be regarded as a specific form of gangrene. Among the bacteria capable of producing a primary gangrene may be mentioned the *Bacillus aërogenes capsulatus*, *B. œdematis maligni*, *B. diphtheriæ*, *B. anthracis*, *B. coli communis*, and probably a number of other bacteria as yet not well known. Secondary gangrene may be caused by burns, freezing, deprivation of the blood supply, mechanical injury, pressure, corrosive poisons, various intoxications, and infections. In all these cases there is first a tissue lesion and inflammatory reaction, associated with or followed by secondary infection with putrefactive organisms. Two chief forms of gangrene are recognized—*dry* and *moist* gangrene. The former occurs in parts exposed to the air and therefore after necrosis quickly losing their water through evaporation. When evaporation does not occur, the parts remain moist and present a better soil for the growth of saprophytic organisms. In the dry form there is usually but little bacterial growth and consequently less decomposition; in the moist form, on the other hand, the putrefactive processes constitute the chief feature. Between the two forms there is no hard-and-fast line. A moist gangrene may become converted into a dry form by evaporation of the fluid contained in the necrotic area, while a dry gangrene, through the absorption of fluids from the surrounding tissues, may become changed into the moist.

According to its origin, gangrene is also classed as *traumatic, thermal, toxic, senile, idiopathic, diabetic, neuropathic,* etc. According to the character of the process, there may be distinguished such forms as *circumscribed, diffuse, phagedenic,* etc. When the putrefaction is very marked, the gangrene is designated

septic or *putrid* gangrene. The formation of gas in the gangrenous area gives rise to *emphysematous gangrene.* When the tissues contain much blood before death, they are usually black or greenish in color (*black gangrene*); when anæmic, they are lighter in color, although always discolored to some extent (*white gangrene*). Clinically these forms are also known as *warm* and *cold* gangrene, respectively.

Dry gangrene is usually circumscribed. It occurs in the parts of the body most exposed to evaporation, as the tips of the ears, nose, fingers, and toes. In the great majority of cases it is due to arterial or venous obstruction by thrombosis or embolism, whenever the collateral circulation is insufficient to keep up the nutrition of the part whose vessels are affected. It also occurs after freezing, burns, corrosions, in ergotism, diabetes, senility, Raynaud's disease, etc. The affected part is discolored, yellow, brownish, or black, or the tissues may at first appear bloodless and very pale. The consistence gradually becomes hard and tough, and finally the part comes to resemble leather or the skin of a mummy. A formation of vesicles or blebs may precede the mummification. Should these rupture and the corium become denuded, the process of evaporation is aided. In the early stages the odor of putrefaction is present, but is never very marked. Around the dead area there is usually present a more or less sharply marked line of demarcation.

Moist gangrene occurs very frequently after severe traumatism of the extremities, obstruction of the arterial or venous flow, in certain skin diseases, diabetes, senility, acute infections, etc. *Decubitus, noma, malum perforans, hysterical gangrene* are varieties of the moist form of gangrene. Of the internal organs the lungs are most frequently the seat of moist gangrene. It follows infarction, inspiration pneumonia, non-resolution of croupous and purulent pneumonia, pulmonary abscess, atelectasis, bronchiectasis, neoplasms, etc. Moist gangrene of the mesentery follows mesenteric infarction due to embolism or thrombosis of the mesenteric arteries. Strangulated hernia, intestinal obstruction, intussusception, obstruction or strangulation of the appendix, traumatic injury of the pancreas, pancreatic inflammation, torsion of the pedicle of new growths, floating spleen, kidney, etc., usually lead to moist gangrene. Extreme passive congestion, marked œdema, infiltration of

Fig. 48.—Gangrene of Portion of Foot. *N*, Necrosed portion; *G*, zone of demarcating proliferations; *H*, living tissue outside zone of demarcation. (*After Ribbert.*)

the perineal tissues with urine, retention of the urine, etc., are also conditions favoring the occurrence of moist gangrene.

Moist gangrene is recognizable clinically by its foul odor, discoloration, and progressive softening of the affected area. In the early stages the color is usually reddish purple, but later becomes greenish, brown, or black. Blebs filled with a dirty brown fluid are formed in the skin of the gangrenous area. Ultimately the entire part becomes soft and partially liquefied, and the phenomena of putrefaction are presented, as in the case of a dead body. Hydrogen sulphide, ammonia, indol, skatol, fatty acids, amins, carbonic acid, and other gases are formed during the putrefactive process. When the gas formation is marked, a local or widespread emphysema may be produced. Around the gangrenous part there is usually a zone of inflammatory demarcation (see Fig. 48). Finally, the dead tissues are either cast off or are absorbed, calcified, organized, or encysted.

Besides the end products of decomposition mentioned above, there are formed in the gangrenous tissue diffusible poisons, which when absorbed produce systemic symptoms. The intensity of the latter depends upon the amount and character of the poisons produced, the amount and rate of the absorption, and the resistance of the patient. The intoxication may be so intense as to cause death. In other cases the gangrenous process advances until death is brought about by the involvement of vital parts. Healing may occur after sequestration, organization, or calcification.

The varieties of gangrene met with in surgical work are numerous. *Senile, diabetic, infective, traumatic, toxic, multiple, neuropathic, hospital, emphysematous, X-ray, carbolic-acid, decubitus,* etc., are among the most important clinical varieties. These will be discussed more fully under the proper heading.

The treatment of gangrenous inflammations is, in general, the same for all varieties. In the infective cases the treatment should be directed along antiseptic lines. In the case of obstruction to the blood supply, exercise, massage, and hot baths may be used to encourage a collateral circulation and to prevent the occurrence of gangrene. The general symptoms should be treated according to indications. As the subject of gangrene from the clinical standpoint will be treated fully in a later article, it will not be necessary for me to enter into further details in this place.

III. CHRONIC INFLAMMATIONS.

Etiology.—The causes of chronic inflammation are to be sought in factors that excite a progressive tissue lesion and in those that prevent prompt healing. Persistent infection, chronic intoxications, repeated injury by extrinsic agents (dust, repeated rubbing, foreign bodies, etc.), unfavorable nutritive conditions, diminished resistance, extensive tissue defects, presence of large masses of ne-

crotic tissue, collections of exudate that are with difficulty removed, etc., are the chief causes of the persistence of an inflammatory reaction.

Chronic infections very frequently give rise to progressive inflammations, which spread through the body by direct extension and give rise to metastases through the blood or lymph. Such chronic inflammations are caused by bacteria and certain moulds and yeasts, which continue to multiply in the body and constantly to give rise to new tissue-irritation and injury. To this class belong chiefly the so-called specific infections, tuberculosis, syphilis, leprosy, actinomycosis, blastomycosis, etc.; but persistent infections due to the ordinary pyogenic bacteria, colon bacillus, typhoid bacillus, gonococcus, pneumococcus, etc., are not infrequent. Such inflammations present a more or less distinct clinical course and symptomatology.

Chronic intoxications play a very important rôle in the production of chronic inflammations of the internal organs, particularly of the liver and kidneys. The chief source of the poisons is to be sought in the gastro-intestinal tract, but substances harmful to the organism may be taken in through the respiratory tract, skin, etc. In many of these chronic inflammations of the internal organs the exciting factor is probably an *auto-intoxication*, the poisonous substances being produced within the body itself as the result of disturbed metabolism, perverted gland function, or failure of specific internal secretions (auto-intoxications).

Repeated mechanical injury, though of slight degree, may give rise to a persistent inflammatory reaction. The repeated inhalation of irritating dusts excites a chronic pneumonia; repeated friction causes inflammations of the skin or mucous membranes; pathological changes in the contents of the gastro-intestinal tract may give rise to chronic inflammations of the mucosa of this tract. The presence of concretions, foreign bodies, etc., likewise causes chronic irritation and a persistent reaction.

Portions of *necrotic tissues* too large to be easily replaced or removed, or that are absorbable with difficulty, act as foreign bodies and excite chronic irritation and reaction. Likewise masses of *purulent* or *fibrinous exudates* act in the same manner, and the reaction persists until the exudate is completely organized, encapsulated, or calcified. Large pieces of necrotic bone may persist as sequestra for a number of years and keep up a constant inflammation. Further, large tissue defects, such as extensive burns and ulcers, require often many months before the wound surface is covered over with epithelium and the healing process completed.

Unfavorable nutritive conditions, such as general or local anæmia, chronic passive congestion, delay the course of healing and also predispose to inflammatory conditions, in that they permit slight extrinsic agents, which ordinarily produce no tissue lesions or at least only slight ones, to set up ulcerative inflammations that show little tendency to heal.

In general, chronic inflammations are characterized by *hyperplasia of the connective tissues* of the affected part. Chronic inflammations of the serous membranes, caused by chronic infection or by the presence of exudates not easily removed or absorbed, are characterized by extensive thickenings of the membrane, due to the organization of the exudate or to a hyperplasia of the connective tissue of the subserosa. (See Fig. 49.) The new formation of connective tissue, therefore, takes place either upon the serous membrane or within it. Dense hyaline adhesions and thickenings may thus result, and the capsule of such organs as the spleen and liver may be enormously thickened ("Zuckergussleber," etc.). Chronic infective inflammations of the lung, prolonged irritation due to the inhalation of dusts, etc., cause a diffuse fibroid induration of the pulmonary tissue (chronic fibroid pneumonia, stonecutter's lung, etc.). A persistent flow of irritating secretions, as in the case of a chronic gonorrhœa, produces a marked hyperplasia of the mucous membrane and skin in the neighborhood of the genital orifices, affecting chiefly the papillæ and overlying epithelium, so that verrucose growths are produced (*condylomata acuminata*). Diffuse hyperplasias of the connective tissue of the skin, known as *elephantiasis*, may be caused by a persistent or frequently repeated trauma, the presence of parasites, etc. Chronic inflammations of the periosteum and bone marrow give rise to new formations of bone. In the case of the internal

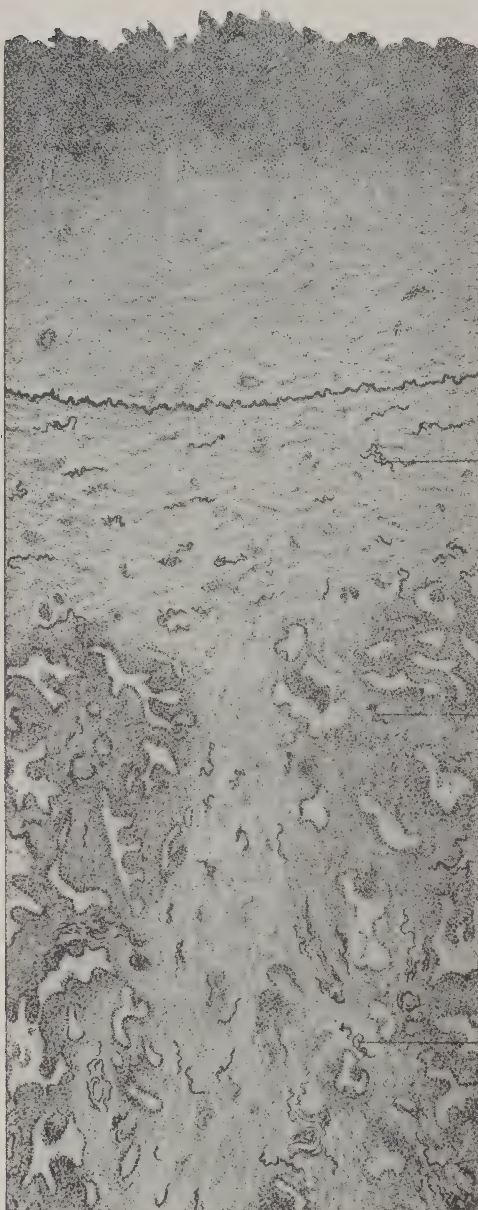

FIG. 49.—Changes in the Pleura and Lung after a Purulent Pleuritis Lasting Six Months (alcohol, orcein). *a*, Thickened lung tissue with gland-like alveoli, and elastic fibres in the newly formed connective tissue; *b*, thickened pleura; *c*, newly formed connective tissue without elastic fibres; *d*, granulation tissue covered with pus; *e*, elastic limiting membrane of the pleura; *f*, elastic fibres. × 46. (*After Ziegler.*)

organs, chronic inflammatory processes are characterized by a local or diffuse hyperplasia of the connective-tissue stroma (hepatic cirrhosis, etc.).

Chronic inflammations may be classed according to their causes or to the character of the changes produced in the affected part. The chief forms are *chronic catarrhs, chronic abscesses, chronic ulcers,* the *infective granulomata, hyperplastic chronic inflammations,* and *atrophic chronic inflammations.*

Chronic catarrhs of the mucous membranes may be caused by prolonged circulatory disturbances (chronic passive congestion), chemical irritation resulting from pathological changes in the gastric or intestinal contents, chronic infections (gonorrhœa, tuberculosis, etc.), the presence of animal parasites, concretions, etc. Chronic catarrhs of the genito-urinary tract are the forms most frequently falling into the province of surgery.

Chronic abscesses may result from acute abscesses and are due to the same causes. In other cases they are the result of a specific infection (tuberculosis, actinomycosis, etc.), and develop more gradually without passing through any well-defined acute stage. Chronic abscesses have a connective-tissue wall lined with granulation tissue. They may contain pus or a material resembling pus, formed by the partial liquefaction of caseous material ("*cold abscess*"). The specific forms of chronic abscesses are as a rule easily distinguished from other forms by the peculiar character of the granulation tissue of the abscess wall and also by the presence of the infective agent. Chronic abscesses increase in size as the result of a progressive destruction of the abscess wall and the neighboring tissue as well as by the continued formation of pus. As they increase in size extension is usually governed by gravity and the path of least resistance. When enlarging progressively toward the deeper tissues, they are spoken of as "*congestive*" or "*burrowing*" abscesses. The increase in size usually indicates the persistence of the infective agent. "*Cold abscesses*" are seen most frequently in cases of tuberculous arthritis and osteomyelitis. The contents of the cavity are, as a rule, slowly formed. "Cold abscesses" of the vertebræ extend downward along the spine and psoas muscles, presenting themselves as fluctuating tumors either above or below Poupart's ligament. In the great majority of cases the chronic abscess is tuberculous. Chronic abscesses also occur as sequelæ of typhoid fever, influenza, variola, and others of the acute infectious diseases.

Chronic ulcers (see Fig. 50) may be caused by non-specific injurious agents, the ulcer for a number of reasons not healing and consequently running a chronic course. Such ulcers are usually found on the inner side of the lower third of the leg, and are generally associated with a chronic congestion or a varicose condition of the veins of the part. In the stomach the healing of an ulcer may be prevented or delayed by peculiar qualities of the stomach contents. In the majority of cases chronic ulcers are the result of some specific infection (*tuberculosis, syphilis, glanders,* etc.). Chronic ulcers vary

greatly in size, shape, appearance of base, edges, surrounding tissues, and general characteristics of the process. A very great variety of terms is, in consequence, applied to them. When extending around a limb they are called *annular ulcers*. If healing begins at one edge of the ulcer while the ulceration advances at other parts, the resulting variety is known as *serpiginous*. A small, slowly progressive ulcer is called an *indolent* ulcer. When pale, soft, and flabby, the ulcer is designated *weak* or *œdematous*. A round or irregular funnel-shaped ulcer is styled *perforating* ulcer. When covered with a grayish or yellowish-white necrotic layer composed of fibrin and necrotic cells, the ulcer may be classed as a *croupous* or *diphtheritic* ulcer. *Raw* ulcers are those in which the base of the ulcer is composed of the body tissues, muscle or connective tissue. A dense, callous thickening of the edge and base of the ulcer

FIG. 50.—Floor of Chronic Ulcer. *a*, Superficial layer of purulent exudate; *b*, zone of chronic granulation tissue; *c*, tissue at base of ulcer. (*After Weichselbaum.*)

gives origin to the form known as *callous* ulcer. Excessive formation of granulation tissue produces an *ulcus elevatum hypertrophicum*. The base of an ulcer may be either lower or higher than the surrounding tissue; it may be pigmented. The edges may be sharply outlined or irregular, thin or thick, adherent, overhanging, rounded, elevated, undermined, "worm-eaten," etc. They are usually of a uniform height. The tissue about a chronic ulcer may be red, swollen, œdematous, pigmented, eczematous, etc. Smaller ulcers may be grouped about the main one. There is usually only a small amount of discharge from a chronic ulcer. The symptoms are much less intense than in the case of an acute ulceration, except when nerves are directly involved.

Among the clinical varieties of chronic ulcer seen in surgical practice are the *varicose* ulcers, *erethistic, perforating,* etc. The *varicose* ulcer is found on the lower or middle third of the leg, in association with varicose veins. It is usually irregular, but after a time becomes more round. The edges of the varicose ulcer are undermined, bluish, pigmented, and show slight granulation; when older, the edges are usually callous. The discharge from a chronic ulcer is, as a rule, small in amount, serous in character, and containing tissue débris and blood. About large varicose ulcers the skin is usually more œdematous, and is not rarely deeply pigmented. Eczema is a frequent complication. The development of granulation tissue is slow and limited in extent. Varicose ulcers may be of small size or may involve the greater portion of the limb. They are often multiple.

The *erethistic* ulcer is a chronic ulcer found over the inner malleolus, particularly in women who have varicose veins or suffer from disorders of menstruation. It is also known as the *congested, irritable,* or *painful* ulcer. Beginning as a small area of hyperæmia over the inner malleolus, it gradually increases in size, becomes more painful, and finally, as the result of some slight injury, develops into an ulcer having sharply cut edges and tightly adherent base. The skin about the ulcer is thickened, pigmented, and adherent. An eczematous condition is also often present. These ulcers are characterized by intense pain, due to the involvement of terminal nerves in the indurated tissue of the base and edges. They are very slow of healing and often return.

The *perforating* ulcer is most often found on the sole of the foot over the head of the metatarsal bones, but may also be found on the fingers or toes. It occurs chiefly in males past the age of forty, and is associated with constitutional conditions, such as syphilis, diabetes, tabes dorsalis, arteriosclerosis, etc. Sclerotic changes or thrombosis of the plantar vessels are probably the direct etiological factors in the majority of cases. The perforating ulcer usually begins as a small callus over the head of one or more of the metatarsal bones. Beneath the callus the abscess develops. As there is usually an accompanying impairment of sensation, the condition is neglected until the bone has become involved. There is finally formed a painless, funnel-shaped ulcer, extending to the bone. In old ulcers of this kind the epithelium may extend over the edges and partly or entirely cover the sides of the funnel-shaped depression. The discharge persists as a scanty, foul, purulent fluid, containing fragments of dead tissue and necrotic bone.

Chronic ulcers may also occur in gout as the result of infection of tophi (*gouty ulcer*). Ulcers of the skin are of frequent occurrence in diabetics and show a tendency to spread rather than to heal. In *syphilis* secondary and tertiary ulcerative lesions are very common on the skin and mucous membranes. They may be superficial or deep, and arise from the breaking down of superficial or deep gummata. They are often serpiginous. The superficial syphilitic ulcers

are usually circular, with sharply cut edges and an indurated base covered with a yellowish, tenacious, purulent exudate. The deeper ones are irregular, with ragged, undermined edges and indurated, sloughing base. *Tuberculous* ulcers occur in the skin and mucous membranes, being of more frequent occurrence in the latter situation. They are usually secondary to chronic tuberculous processes in other parts of the body, and are caused by the caseation of local tubercles with secondary infection. The edges of the tuberculous ulcers are usually elevated, indurated, and not undermined, and the base is made up of pale, caseous, tuberculous granulation tissue. The tuberculous ulcers of the skin appear in a variety of forms clinically, and have been designated according to their most prominent characteristics (*verrucose, indurated, scrofuloderm,* etc.). Actinomycosis, blastomycosis, leprosy, and other of the specific infections often give rise to chronic ulcerative processes.

More or less atypical formations of granulation tissue characterize various specific infections (tuberculosis, syphilis, leprosy, rhinoscleroma, actinomycosis, blastomycosis, glanders, etc.). They form nodular or diffuse chronic prolifera-

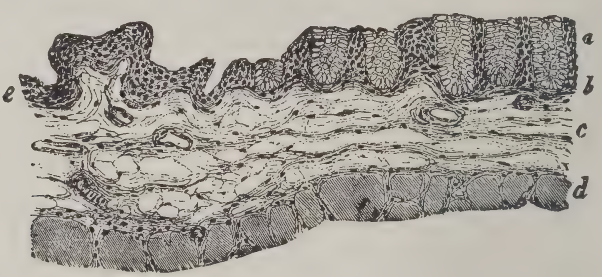

FIG. 51.—Chronic Atrophic Colitis. *a,* Atrophic mucosa, *b,* muscularis mucosæ; *c,* submucosa; *d,* muscularis; *e,* total atrophy of mucosa. (*After Ziegler.*)

tions of granulation tissue, which are classed under the general heading of *infective granulomata.* As a rule, they possess more or less well-defined characteristics of structure and course, that permit of a clinical and pathological diagnosis. The presence of the etiological factor also aids in the differential diagnosis. The etiology of some of the granulomata is, however, still unknown. Various clinical designations are applied to different forms of granulomatous conditions (*fungous granulations, caro luxurians,* etc.).

Chronic inflammations characterized by marked *atrophy* of the parenchymatous tissues, with or without hyperplasia of the connective tissue, are usually called *atrophic.* (See Fig. 51.) They occur particularly upon the mucous membranes of the gastro-intestinal tract and the bladder, and in the liver and kidneys. In the gastro-intestinal tract the epithelium becomes atrophic as the result of persistent desquamation or necrosis, the connective tissue either being unaffected or undergoing necrosis at the same time. It rarely shows marked hyperplasia in this location. In the case of chronic inflammations of the liver and kidneys, the atrophy and necrosis are accompanied or followed by a

more or less marked hyperplasia of the connective-tissue stroma (hepatic cirrhosis, contracted kidney). In the case of the liver, imperfect attempts at regeneration of liver tissue lead to a hyperplasia of the small bile ducts. (See Fig. 52.) Such chronic inflammations are known as *productive, indurative, hyperplastic,* etc.

The treatment of chronic inflammations is both constitutional and local.

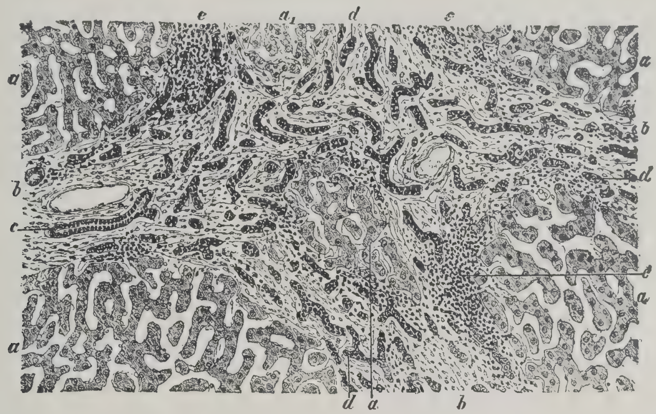

Fig. 52.—Connective-tissue Hyperplasia and Proliferation of Bile Ducts in Chronic Hepatitis (alcohol, hæmatoxylin). *a*, a_1, Liver lobules; *b*, hyperplastic periportal connective tissue; *c*, old bile ducts; *d*, newly formed bile ducts; *e*, foci of small-celled infiltration. × 55. (*After Ziegler.*)

The general condition should be built up and improved by proper food, hygiene, tonics, etc. The cause of the chronic reaction must be removed or inhibited when possible. Dead tissue, exudates, foreign bodies, etc., should be sought for and removed. The general indications for the treatment of inflammatory conditions—rest, absolute cleanliness, asepsis, etc.—should be met. The formation of granulation tissue should be stimulated in the various ways mentioned above, plastic operations and skin grafting carried out when necessary, and the promotion of cicatrization and new epithelial growth encouraged.

THE NATURE AND SIGNIFICANCE OF THE VARIOUS DISTURBANCES OF NUTRITION OBSERVED IN CONNECTION WITH SURGICAL DISEASES AND CONDITIONS.

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INTRODUCTORY.

Before we can attain anything like a proper comprehension of the processes with which we are to deal in the following pages, we must have some knowledge of the general principles underlying the question of the growth and development of organized structures. All those physical and chemical phenomena included under the term "nutrition," which are so peculiarly the attributes of living substance, can be understood only by a reference to the fundamental properties of protoplasm.

Every living organism, whether animal or vegetable, must be regarded as being composed of one or more cells. All but the lowest forms of life are essentially aggregations of cells or communities of primordial units. The highest and most complicated individuals, whether plant or animal, are composed of organs, these of tissues, and these again of cells. Ultimately, then, the life history of any individual is the sum total of the life histories of its primitive constituents. The laws which govern the cell are those which govern the individual as a whole.

Every organized being begins as a single cell—the fertilized ovum. This cell is peculiar, in that it represents in a large measure the characteristic tendencies and properties of the parents. What it will become depends not only on the specific characters implanted in it, peculiarities which we speak of as *inherited,* but also on the influence of external forces, or *environment.*

The first step in the development of the complete individual is the division of the ovum. The resulting cells increase in size, owing to the influence of the pabulum supplied, and finally become specialized according to the function which they have eventually to perform. We can thus recognize three fundamental attributes of primitive cells: (1) Their capacity for multiplication; (2) their power to increase in size; and (3) their tendency toward histological differentiation.

The term "growth" implies an increase in size of the organism. This increase is, however, not true growth, unless it be the result of forces inherent in the cells. Enlargement of an organ or tissue may be due to the deposit within

it of some product of degeneration, such as fat, or to a variety of other extrinsic causes. This is not growth. The growth of the individual is the expression of the growth of his component cells. Growth, then, is ultimately dependent on an increase in the size of single cells and on a multiplication of their numbers. Growth, however, implies somewhat more than this, in a vague way, namely, the power of the cellular elements to advance in the scale of organization, or at least not to retrograde.

The three factors just referred to—multiplication, increase in size, and histological differentiation—so far as the organs and tissues are concerned, operate in harmony and to some extent coincidently, but with regard to the individual cells are more or less mutually exclusive. Cells which are rapidly dividing are always small, since they have no resting period in which to increase their bulk. The finer details of histological differentiation form the last stage in the development of any structure. Conversely, cells which are highly specialized lose to a large degree the power of multiplication, or, if they do multiply, must first revert to a more primitive condition.

Development is in a sense a thing apart from growth, for a cell or tissue may attain practically its full size, in proportion to the individual concerned, without attaining its highest degree of specialization. Generally, however, we employ the term "development" in a broad way, to include not only growth in size, but also differentiation.

The capacity for growth and development possessed by all cells and tissues is not everywhere manifested in the same degree. Certain structures may take years to attain their anatomical and functional completeness, while others quickly mature and as quickly retrograde.

Let us examine a little more carefully the question of multiplication of cells and the increase in size of tissues.

Believing, as we do, in the doctrine that *every cell is derived from a previously existing cell*, we are apt to go somewhat further and assume that one kind of cell invariably gives rise to one of the same type; for example, that epithelium gives rise to epithelium, muscle to muscle, nerve cell to nerve cell, and so on. This is not necessarily so, as a little reflection will show us. The mere fact that a single, undifferentiated cell, the fertilized ovum, is the precursor of all the cells of the body and gives rise to the most highly specialized structures, such as the retina, might have prepared us for something different. As a matter of fact, when we study growing tissues we see that certain cells alone are actively dividing or are actively proliferative, while others do not divide, but assume other special functions. If we take the case of a growing plant, we find that at the tip of each stem or rootlet there is a mass of cells of embryonic type, which are undergoing rapid division. Development takes place in a very peculiar way. At the extreme end of the stem is a cell or group of cells, that always divide by transverse fission into a distal and a proximal daughter cell or cells. The distal

daughter cell at the tip always retains its embryonic characters and keeps on dividing in the same manner. Thus, the original proximal daughter cell becomes separated from the tip of the stem by successive divisions of the apical cell or cluster of cells. The distal cells never do anything but divide. The proximal ones, however, have another destiny. They eventually cease multiplying, become larger and vacuolated, and eventually assume the characters of the cells of the tissues to which they belong. In other words, they become differentiated. Thus we see that certain cells are specialized for multiplication, others for growth and for histological differentiation. The same thing holds in connection with animal life. The more highly differentiated cells of a tissue do not give rise directly and by division to other cells of the same type, but in each tissue there are undifferentiated cells—mother cells, or embryonic cells—which have the special power of proliferating; and it is the daughter cells, derived from these, that reach the highest anatomical and functional perfection.

Proofs of this might be multiplied. As a consequence, we may lay down the following general principles: 1. That fully differentiated cells of a tissue never give rise directly to cells of the same highly specialized type. 2. That in all tissues there are certain "embryonic" or undifferentiated cells, whose special function is to divide, and that the daughter cells derived from these are the ones which subsequently attain the higher planes of development. 3. The more highly specialized the cell, the more difficult it is for it to reproduce its kind; conversely, the more primitive the type of the cell, the more easily will it proliferate. These general laws, which hold good in regard to the natural growth and development of the organism, are equally true in connection with pathological processes. Injured or lost parts are restored to the normal in accordance with the same laws which governed their formation in the first instance. As the higher forms of cell are derived from the lower, so must they revert to a more embryonic condition before they can undergo proliferation.

Finally, as we have three fundamental properties of protoplasm, so we have three methods by which its vital energies are manifested—assimilation, nutrition, and reproduction. Any interference with these will cause disease.

HYPERTROPHY.

In a general way, hypertrophy may be defined as an increase in the substance of a tissue or organ, due to an increase in the number or in the size of its component cells, or to a combination of both these conditions, without any other alteration of structure. Like many other definitions, this one needs to be defined.

As we have seen, growth consists in the increase of tissue by the addition of new material of the same nature as that already existing. The essential character of the part is not altered, but its bulk is increased, and it is therefore en-

abled to perform more work than it had previously been doing. Up to a certain point development and growth go hand-in-hand. When the time comes that all the necessary parts of a structure are complete, development ceases, although growth may for a time continue until the perfect stature and proportion of the body are attained. It is not, however, possible to fix the point at which either development or growth ceases, for in this particular different organs and structures have their own peculiarities, and much depends on personal idiosyncrasy and the condition of the bodily health. Some organs, like the thymus, reach their full perfection during childhood, and early atrophy and disappear; others, like the heart and arteries, have been shown to go on receiving increments of substance and power until advanced years. Much, however, depends upon circumstances. If the bodily health be robust and the organ in question be regularly and fully exercised, then growth will continue, or, at all events, the organ will not waste. Any addition to the amount of work demanded of any organ will result in an increase in its size. To a certain extent this is desirable and can hardly be regarded as an evidence of disease. In fact, it is often difficult to draw the line between what may be termed physiological hypertrophies and the pathological overgrowths, for both are essentially conservative processes, tending to maintain the nutrition and function of the part in the highest possible efficiency. It would appear, too, as if all organs possessed, though in varying measure, a certain reserve power of growth and development, which they are able to put into action in case of necessity. In this way the extra demand is compensated.

The orderly and natural progression of the metabolic processes, which is the indication of health, is dependent upon the correlation of several factors, the vascular supply of the part, its innervation, and the mutual relationship with other structures. There is, so to speak, a constant effort toward an equilibrium. In the case of overgrowth the same general laws are at work. The possibility of the occurrence of hypertrophy, and its efficiency when it has arisen, depend essentially upon the state of nutrition of the affected part. This implies an adequate vascular and nervous supply. The blood-vessels must therefore enlarge and the trophic centres be correspondingly active. The blood also must be of good quality. Once the reserve power of the part is exhausted, not only does the process of hypertrophy cease, but degeneration and atrophy take its place, resulting in functional inadequacy of the structures concerned. Consequently, as we would expect, we get the most extreme examples of hypertrophy in the young and robust, while, on the other hand, in the aged and debilitated the ordinary causes will fail more or less completely to produce it.

Not only, however, does the term "hypertrophy" connote an increase in size, but there may be, in addition, a formation of new tissue. Thus, in the hypertrophied heart the muscle fibres are more numerous, stronger, and more highly colored. In the liver of atrophic cirrhosis, while many of the parenchymatous cells are wasted, fatty, and degenerated, new ones are formed, which are

almost gigantic, having large, deeply-staining nuclei. In the pregnant uterus new and relatively large and powerful fibres are produced. It is usual, therefore, to recognize two forms of hypertrophy. Where there is a simple increase in the size of the cells composing the part, we speak of *quantitative* or *true hypertrophy;* if there be an increase in the number of the cells, we speak of *numerical hypertrophy* or *hyperplasia.* As a rule, both conditions are combined.

According to the nature of the cause at work, hypertrophy may be temporary or permanent. Many of the temporary hypertrophies are physiological in their nature, as, for instance, the enlargement of the uterus during gestation, the enlargement of the muscles from increased exercise, the production of new and active acini in the breast during pregnancy as a preparation for lactation. Should the cause persist or from its nature be irremovable, temporary hypertrophy becomes permanent and may lead to important consequences.

As a rule, it is possible to discover some reason for the production of hypertrophy in a given case. There are, however, instances which are more or less obscure. Such are the hypertrophies which are occasionally seen in the thyroid and thymus glands, the spleen, tonsils, and prostate, and in warts and polypi. Some, possibly, may have an inflammatory basis, while in the case of the ductless glands there may be some disturbance in a correlated organ. But more than this we can hardly say, in view of the present state of our knowledge, or, rather, want of knowledge. We must for the time being be content to recognize certain hypertrophies, which have affinities, on the one hand, with inflammation, and, on the other, with neoplastic growth.

The anatomical changes peculiar to hypertrophy are usually, though by no means invariably, manifested by an increase in the size and weight of the affected structure. Mere increase in weight does not, however, constitute hypertrophy. For example, the heart may be enormously enlarged in point of its external configuration, owing to dilatation of its cavities or from a deposit of fat upon its surface; the liver may be enlarged from hyperæmia or inflammatory infiltration. Conversely, the heart may present no external evidences of hypertrophy, in that its bulk is not increased, and yet it may be truly hypertrophied. This occurs in the so-called "concentric" hypertrophy, in which the enlarged muscle has encroached upon the cavities. Before, then, either increased size or weight of an organ can be taken as an evidence of hypertrophy, we must make sure that this increase is not due to any cause but the increase in size or number of the pre-existing cells of the part. This, as a rule, can be determined only by a careful microscopical examination.

Histologically, true hypertrophy is indicated by an increase in the bulk of the individual cells. The nucleus is enlarged, often altered in shape, and stains more intensely than usual. Thus, in the case of the heart muscle, the nucleus of the fibre is enlarged and more obtuse at the poles than normal. Nuclear division is not infrequent. The cell body is also enlarged, and the cytoplasm shows an

increased affinity for stains, such as eosin, being of greater intensity and brilliancy than that of similar cells under normal conditions. In the case of pigmented cells, such as those of the muscle, the pigment appears to be increased. These minute changes can be properly appreciated only by a comparison with the normal condition of things and by accurate measurements of the cells, although an expert microscopist will usually be able to reach fairly correct conclusions without such assistance.

Rarely or never do all the cells of an organ or tissue manifest the same degree of hypertrophy throughout. For, paradoxical as it may seem, hypertrophy and atrophy often go together. The cells may be increased in size but diminished in number, or, conversely, hyperplasia may be accompanied by a decrease in bulk of the individual elements.

Granting, then, a natural tendency of cells toward growth, which, under certain circumstances, may be in excess of the normal, we find that the causes at work in the production of hypertrophy are either *intrinsic* or *extrinsic.* In the former case the abnormal tendencies to cell growth and multiplication appear to be inherent in the cells, the result of some peculiarity in the germinal cells or of germ variation. Here the anomaly is present at birth or makes its appearance comparatively soon after. Extrinsic hypertrophy is usually the result of increased nutrition and excessive demand upon the function of an organ, or of a disturbance of the equilibrium that ought to subsist between anabolism and katabolism.

Intrinsic Hypertrophy.—This is congenital or else appears shortly after birth. As a rule, the internal viscera are not involved, unless we accept certain obscure enlargements of the brain, thymus, spleen, and lymph nodes as of congenital origin. The condition may be universal or partial. A number of organs and tissues may be affected. Hypertrophy of the epidermis gives rise to the condition known as *congenital ichthyosis.* Increase in the amount of hair, or its appearance on parts of the body that are normally destitute of it, is called *hypertrichosis.* Enlargement of the nails is *hyperonychia.* The amount of fat may be excessive—*lipomatosis* or *obesity. Elephantiasis* is a term used somewhat loosely to designate a number of conditions which have this in common, that the affected part is enlarged (see Fig. 53). The tropical form of elephantiasis is not a true hypertrophy, but is more akin to inflammation. The enlargement is due to the obstruction of the lymphatics, with secondary hyperplasia of the connective tissue. There are a number of congenital conditions, however—as, for instance, certain nævi and enlargements of the face, lips (*macrocheilia*), and tongue *macroglossia*)—which appear to depend on some obstruction of the lymphatics and blood-vessels, with, in some cases, apparently actual new-formation of vessels, which possibly may be included under hypertrophy.

Perhaps the best example of congenital hypertrophy is *gigantism.* This may affect the body as a whole—bones, muscles, skin, nerves, vessels, and internal

organs (*general gigantism*)—or some particular organ or member (*partial gigantism*).

In *true* or *essential* gigantism the individual affected differs in no respect from the normal, save in the one particular of size and weight. In addition to excessive size and weight, there are great strength and perfect proportion, together

Fig. 53.—Elephantiasis of the Leg; Enormous Enlargement of the Limb, with Ichthyosis. (*Pathological Museum, McGill University.*)

with ordinary intelligence. Such giants are examples of the so-called "athletic" habit of body, and represent the human body carried to its highest power in point of structure. Cases of this kind have no doubt existed, but are excessively rare. As a rule, however, giants present the unmistakable stigmata of defect. The increase in height is due mainly to excess in the long bones; the head

is proportionately small; there are evidences of infantilism, knock-knee, and genital inadequacy; and, finally, physical and mental weakness.

Besides this form of general gigantism, or, as it might perhaps be more correctly styled, *macrogenesy*, there are certain less extreme manifestations of the tendency in the form of local hypertrophies, which are undoubtedly of developmental origin. Such are the forms which involve the head or extremities. In *leontiasis ossea* there is an excessive and remarkable deformity of the bones of the face and skull, which appears to be essentially a diffuse hyperostosis. Local gigantism in children is especially common in the upper limbs, and may be unilateral or bilateral. Hemihypertrophy of the body has also been described. One or more digits may be affected (*macrodactylia*), or a whole limb. Apart from these instances of local gigantism, which are characterized mainly by an increase in the bulk of the part, there are certain other forms of numerical increase,

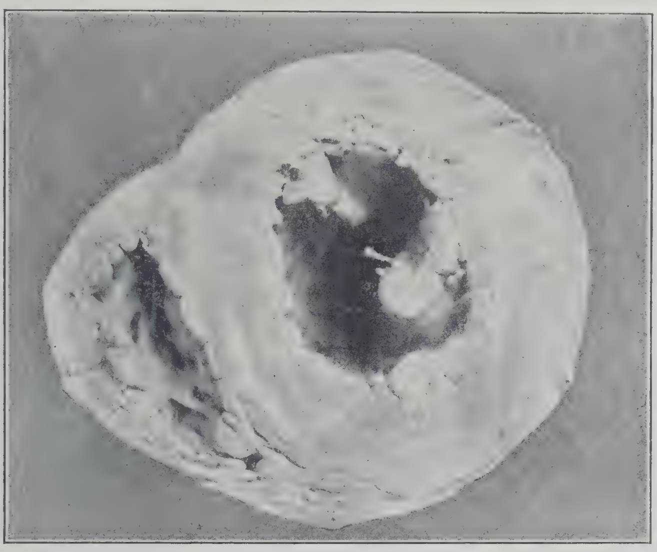

Fig. 54.—Cross Section of the Heart, to Show Hypertrophy of the Walls. The cause: increased peripheral tension. (*Pathological Museum, McGill University.*)

which some authorities would include under the heading "gigantism." Such are polydactylism, accessory ribs, and supernumerary organs. These peculiarities are occasionally associated with general gigantism.

Extrinsic Hypertrophy.—Hypertrophies which are not dependent on some constitutional and inherent peculiarity, but are the result of some external cause, are called *extrinsic hypertrophies*. They are, in other words, *acquired*. It is undoubtedly somewhat difficult, if not impossible, in many cases to draw the line between the congenital and the acquired forms; for some cases, which closely resemble the congenital varieties, may on occasion result from external causes. Such are certain forms of ichthyosis and elephantiasis. It is not impossible that these external causes may have, in some cases, been operative during

intra-uterine existence. It is probable, moreover, that in many instances, even where the condition is evidently due to external influences, there is some inherited tendency to overgrowth of tissue as well. As in the case of cancer, there must be some predisposition of the cells before the exciting cause can wake up the latent activity.

Hypertrophies not dependent on a constitutional idiosyncrasy are always the result of an increased demand upon the functional activity of the part or of some disturbance of the balance which ought to exist between waste and repair. In the majority of instances it is the former. We may recognize the following varieties, to be more precise, namely:

(1) Hypertrophy from increased functional activity.
(2) Hypertrophy from lessened wear.
(3) Hypertrophy from removal of pressure.
(4) Hypertrophy from failure of involution.
(5) Hypertrophy from increased nutrition.
(6) Hypertrophy from chronic irritation.
(7) Hypertrophy from errors of metabolism.
(8) Hypertrophy of neuropathic origin.

Hypertrophy from increased work usually affects the muscles and glands, less often other tissues. In some unexplained way the unwonted physical and chemical condition of the muscle or gland cell leads to excessive cell growth.

Perhaps the best example of this form, which has been called *labor* or *functional* hypertrophy, is to be found in the heart. Increased peripheral vascular tension, certain renal and pulmonary diseases interfering with the circulation, and obstructive valvular affections of the heart itself, lead to overgrowth of that portion which feels the strain most, and eventually to involvement of the whole organ. (See Fig. 54.) Again, as Thoma has shown, increased arterial tension produces hypertrophy of the middle coats of the arteries.

It is a matter of common observation that increased muscular exercise results in increased bulk of the muscles. This is particularly well seen in the case of laborers, in whom certain muscle bundles or groups are often picked out. It is believed that the individual fibres increase not only in length and thickness, but also in number. Many interesting examples are also to be found in the case of the involuntary muscles. Thus, the uterus may be considerably enlarged when the seat of fibroid tumors or of hæmatometra. Any obstruction in the alimentary tract—œsophagus, stomach, or intestines,—whether due to foreign bodies, tumors, adhesions, or strictures, will inevitably lead to overgrowth of the tissues above the obstructed point. The same thing occurs in the urinary bladder in cases of enlarged prostate, stricture, or calculi. In these hollow organs the walls often become enormously ballooned out, but with this there is always an increase in substance. This affects chiefly the muscular layers, but the mucous membrane must

also enlarge to accommodate itself to the changed order of things. In all such cases there is an attempt on the part of nature to overcome by muscular force the hinderance to the proper performance of function. It should, however, be pointed out that mere frequency in the performance of a muscular act is not, so far as we are aware, competent in itself to produce hypertrophy. The heart may, for example, beat more rapidly than normal for years without increasing in bulk and power. The action must at the same time be forcible. This is well illustrated in the case of mechanics. The hand muscles in those who use speed are usually not so large as in those who exert great muscular force. The same thing can be seen in the case of sprinters, athletes, greyhounds, and racehorses, who not infrequently suffer from hypertrophied heart. In all, not only is the heart's action increased in rapidity, but it is excessively forcible. The blood pressure is temporarily raised during the great effort, but the rapidly-acting muscles demand an increased supply of nourishment. The first effect of this is dilatation of the heart, which subsides after the extra call has ceased, but, when such demands are repeated, finally gives place to hypertrophy.

Another form of overwork is seen in some of the secretory organs. Of all glands, the kidney and the liver appear to have the greatest powers of cell growth and proliferation. In diabetics and those who drink to excess the kidneys may be considerably enlarged. Hypertrophy of the liver as a whole is rare, but the liver possesses considerable powers of regeneration, as may be seen in cases where portions of the liver have been removed. The original weight, though not the shape, is quickly restored. Hypertrophy of single cells or small groups of cells is, however, by no means uncommon. This is met with even in such acute conditions as acute yellow atrophy, and is a constant accompaniment of chronic passive congestion and all forms of cirrhosis. The "hobnails" of the gin-drinker's liver are not due, as is so often taught, to the contraction of the fibrous bands, but to an actual hypertrophy and hyperplasia of the parenchymatous cells.

Inasmuch as all such cases of cell proliferation are attempts on the part of the organism to make good or compensate some abnormal condition, these hypertrophies are frequently termed "compensatory." Many of them, as we have seen, are the result of disease in some part of the body more or less remote from the organ or tissue affected. An important class of cases is that which might be termed "complemental." Such are the hypertrophies which occur in one or other of a pair of organs or in structures that are accustomed to work together. Should, for instance, one kidney fail to be properly developed from agenesia or hypoplasia, or should it be at any time removed, or, again, be the subject of some disease that materially interferes with its function, the remaining kidney will attempt to overtake the increased work, and, as a consequence, will hypertrophy. In such cases not only may there be a hypertrophy of the pre-existing structures, but, in young individuals at least, there may be an actual new for-

mation of glomeruli and tubules. The enlargement of the good kidney is usually much greater in the case of congenital deficiency of the organ than in cases of acquired disease—a circumstance which goes to support the principle already indicated, that the power of regeneration is greater in young than in old cells. Similarly, destruction of one suprarenal may be followed by hypertrophy of the other, and agenesia or hypoplasia of one lung may be followed by hypertrophy of the remaining organ. In older persons, however, where one lung is the subject of disease, the compensation takes the form of emphysema rather than that of true hypertrophy, inasmuch as the air sacs dilate—a condition which results in atrophy of the alveolar walls. Here, again, increase in bulk does not indicate hypertrophy.

The power of compensation, which is so strikingly exemplified in the process of hypertrophy, is beautifully illustrated in certain affections of the lower extremity. When, for instance, the tibia is weakened from rarefaction or necrosis or from a badly repaired fracture, the fibula becomes unduly thick and strong in order to meet the increased strain.

A second and perhaps the more interesting class of cases has to do with organs which are functionally complemental. The most notable instance of this is found in connection with the thyroid gland and pituitary body, which are now generally believed to be closely related and of great importance in the bodily metabolism. Loss of function of the thyroid, as from disease or the removal of a portion of the gland, is followed not only by hypertrophy of the remaining part, but by overgrowth of the pituitary. Again, as Warthin has recently shown, in cases of pernicious anæmia and leukæmia, where the function of the bone-marrow appears to be impaired, the hæmolymph glands become enlarged and their structure alters until it comes to resemble that of the spleen or bone-marrow.

Tissues which are normally the subjects of constant wear often attain an abnormal size under conditions where the loss of substance ceases. This generally occurs in connection with the teeth, nails, and skin. The teeth are normally kept at a constant length, owing to the attrition which takes place in consequence of the function of mastication. If the teeth do not properly approximate, as in cases where certain teeth are movable or have fallen out, or, again, in fractures of the jaw, the unopposed teeth gradually elongate and may even form tusks. This is seen normally in certain animals, like the wild boar, and is found occasionally in rodents under the circumstances mentioned. (See Fig. 55.) The tendency for such teeth to grow in a circle is explained by the fact that the enamel of the posterior aspect is more yielding than that of the anterior. In bedridden patients, in whom the ordinary wear is prevented, the nails often become thickened, elongated, and deformed. (See Fig. 56.) Failure to cut the nails is followed by a similar elongation, as may be seen in the nails of the Chinese exquisite or of the Hindu fakir. An analogous overgrowth is occasion-

ally met with in the beak of birds and in the formation of horny pads on the feet of animals.

Hypertrophy also results from the removal of pressure, or, to put it somewhat differently, from a disturbance of the mutual tension which exists between tissues. If from any cause the brain lags behind in its development, the skull remains small in order to accommodate itself to the abnormal condition of things (microcephaly). This, however, occurs only during the developmental period of life. At a later time—that is, after adult age has been attained—any loss of substance of the brain, as from atrophy or disease, is compensated in another way. Theoretically, the wasting of the brain would leave a space between the dura and the calvarium. But, as "nature abhors a vacuum," the space is filled up either with watery fluid (hydrops ex vacuo) or by an overgrowth of the cranial vault. The diploë and the inner table are reconstructed and enlarged, so that the skull-cap may become greatly thickened, although its external appearance may remain unaltered. Such hypertrophy is usually most marked in the neighborhood of the primitive centres of ossification of the cranial bones—a fact which illustrates in an interesting way the uniformity of the law which governs growth and development, whether in normal or in diseased conditions. An analogous condition is the overgrowth of the fat which takes place about a contracted or atrophied kidney, the loss of substance being thus, though in an inadequate fashion so far as function is concerned, made good.

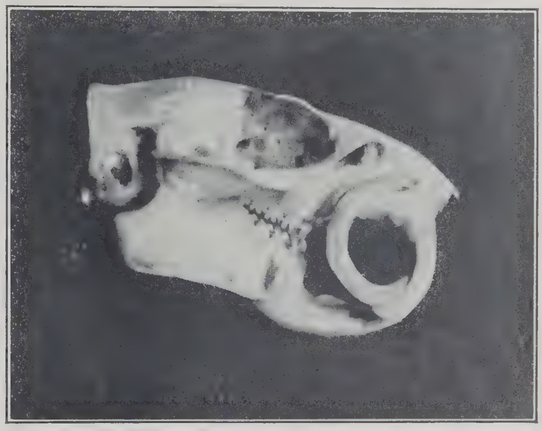

Fig. 55.—Head of a Woodchuck, showing Hypertrophy of the Incisor Teeth from Lessened Wear. (*Pathological Museum, McGill University.*)

Failure of involution to take place results sometimes in a permanent enlargement of the affected organ. The thymus, which attains its full growth and perfection about the second year of life, from that time on begins to atrophy, until at puberty but little of it is left, and about the thirtieth year it is represented merely by fat and connective tissue and some scanty remains. Occasionally the thymus, in its perfect structure, may persist even after puberty. Why this peculiarity occurs and what may be its significance are still matters for inquiry. Another and a common form is the subinvolution of the uterus, which sometimes occurs after delivery.

It has been laid down as a general principle that before growth or overgrowth of a tissue can occur there must be an adequate supply of healthy blood.

For only by means of an active blood supply can the nutrition of a part be kept up. If we examine a hypertrophied tissue we generally find that the blood-vessels supplying it are also enlarged. This is probably in most cases a secondary phenomenon. Here, as in other spheres, the demand creates the supply. But the converse is equally true, that an excessive blood supply frequently leads to hypertrophy. The enlargement of the voluntary muscles from exercise already referred to, is not altogether due to increased function, but in a large measure to the increased degree of nutrition which this implies. For, from the very nature of the mechanical action, the heart is stimulated, an increased amount of blood is determined to the part, and, while the wear and tear are greater than normal, the poisonous products of metabolism are more rapidly eliminated. Apart, however, from mechanical action, many interesting examples may be cited to show the influence of a mere increase in the amount of blood reaching a certain part. The "clubbed fingers" so often seen in chronic pulmonary and cardiac affections seem to be in the main the result of venous hyperæmia. A more marked enlargement affecting both bones and soft tissues is met with in the so-called hypertrophic pulmonary osteoarthropathy of Marie. The cause here is probably the same, aided possibly by the local action of toxins absorbed from the pulmonary lesion. The application of mustard, blistering fluids, or any substance which induces hyperæmia of the skin may at times lead to an increased growth of hair. Similar overgrowths of hair are occasionally met with in the neighborhood of chronic ulcers, about the ends of stumps which have been for a long time inflamed, and about old diseased joints. Sir James Paget records having met with a curious instance of this kind in a child about five years of age. The femur had been fractured near the middle; the case had done badly, and union had taken place with much distortion. The affected thigh was covered with dark hair like that of a strong, coarse-skinned man, while on the rest of the body the hair was as delicate and soft as it usually is in childhood. Cases such as these cannot properly be regarded as the result of inflammation, for the growth of hair is usually at such a distance from the inflamed area as to preclude the

Fig. 56.—Hypertrophied Toe Nails, Removed by Operation. (*Pathological Museum, McGill University.*)

possibility of any morbid influence, save that of hyperæmia alone. This would appear also to be indicated by Hunter's classical experiment of transplanting the spur of a cock upon its comb. The comb is highly vascular, and the transplanted spur reached striking dimensions, being about six inches long and spirally curved. It may possibly be that the congenital hypertrophies known as partial or local gigantism are a consequence of increased vascularity, although this has never been proved.

Closely akin to this last form are the hypertrophies due to chronic irritation. Here an important factor is pressure. Constant pressure usually produces atrophy or necrosis. Intermittent pressure, on the other hand, often leads to hypertrophy. Common instances of this are corns, the calluses on the hands of workingmen, and on the feet. The necessary conditions appear to be a period of stimulation and a period of rest, to allow the processes of nutrition to go on. The effect of this is a hyperplasia and hyperkeratinization of the epidermis, which leads to pressure upon the papillary layer and consequent irritation. Subsequently adaptation may be so far carried out that a secreting bursa may be formed beneath the corn for the protection of the joint.

Fig. 57.—New Growth of Osteophytes about the Hip Joint, the Result of Chronic Arthritis. (*Pathological Museum, McGill University.*)

More interesting still are the cases of elongation and enlargement of bones resulting from inflammation. In inflammation we have not only the influence of an increased flow of blood and increased nutrition, but often also the stimulating effect of toxins, bacterial or metabolic.

Normally the growth of bone depends on the activity of certain specialized cells—the osteoblasts—which are chiefly situated in the deeper layers of the periosteum, at the extremities of the long bones, and at the interosseous sutures. Growth in thickness takes place by subperiosteal osteoplasia; growth in length

is due to the action of the osteoblasts situated at the spongy ends of bones and in the epiphyseal cartilages. Increase in length of a bone depends not only on the inherent vegetative power of the cells, but also on the condition of the epiphyseal discs. Growth in length can occur only so long as the epiphyses are ununited to the shaft. After this takes place, growth in thickness and in density is alone possible. In young individuals, then, irritation in the neighborhood of the ends of a bone may result in increase in its length. Experimentally, in rabbits, the length of the bones can be increased by driving ivory pegs into the epiphyseal discs. The effect of chemical substances is well illustrated also in the experiments of Wegner, who was able, by feeding rabbits for a prolonged period with minute doses of phosphorus, to produce stimulation at the epiphyseal sutures, with consequent increase in the length of the long bones. Arsenic has a similar effect.

Fig. 58.—Femur; Ununited Fracture through the Great Trochanter; Excessive Growth of New Bone. (*Pathological Museum, McGill University.*)

Chronic inflammation affecting a bone or some of the tissues in its neighborhood in a similar way leads to increase of growth. (See Fig. 57.) Thus cases are occasionally met with where, owing to necrosis of some part of the femur, that bone has elongated until the limb was an inch or two longer than its fellow. In such a case the femur does not materially alter its shape or direction. It is different, however, with the tibia. The tibia is bound to the fibula at each end by ligaments, and when it elongates it must necessarily assume a curved position unless the fibula enlarges simultaneously. The stimulating influence of the irritation may, indeed, be traced to a considerable distance from the site of the lesion. Instances are on record where necrosis of the tibia and shortening of the leg have been followed by a compensatory elongation of the femur, so that the limb as a whole was eventually no shorter than the other. Chronic ulcers of the skin and subcutaneous tissues are sometimes followed by elongation

and enlargement of the underlying bone. Similarly, hypertrophy may occur to repair a structural defect. In a badly united fracture the permanent callus is often very large. (See Fig. 58.) It has been shown also, in dogs, that removal of a portion of the radius is followed by an increase in size of that portion of the ulna which is directly opposite to the loss of substance.

Another aspect of the subject should also be referred to. Where the epiphyseal cartilages are united to the shaft of the bone, as in adults, or have been de-

FIG. 59.—Spondylitis Deformans; Curvature of the Spine, with Ankylosis, due to Subperiosteal Osteogenesis. (*Pathological Museum, McGill University.*)

stroyed by disease, increase in length of the affected bone is no longer possible, but increase in its thickness may take place. And, in fact, even in young persons more or less periosteal osteogenesis usually accompanies any increase in length. Increase in the thickness of the bone is a not infrequent event in such affections as chronic, osteitis periostitis, and osteomyelitis. (See Fig. 59.) The overgrowth of the bone is, moreover, not entirely the result of external accretion, due to stimulation of the subperiosteal osteogenetic layer, but to an alteration

of its internal structure. There is, in addition, a deposit of new bone on the trabeculæ, so that the cancellar spaces are obliterated and the texture of the bone becomes more dense and approximates to ivory (*osteosclerosis*). (See Fig. 60.)

As we have hinted above, growth and development are in large measure a question of metabolism. In this connection the internal secretions are of the utmost moment. No doubt all the ductless glands play an important rôle, but

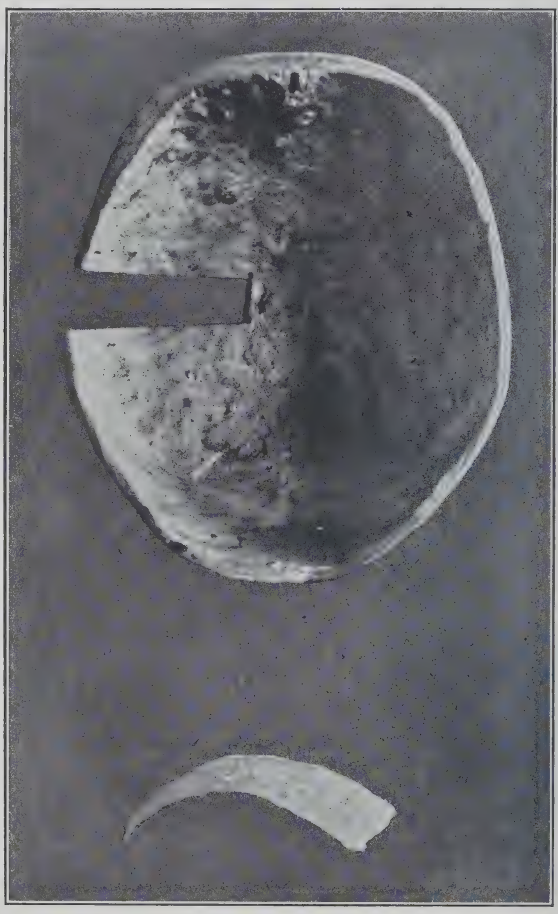

Fig. 60.—Sclerosis of the Calvarium, of Syphilitic Origin. Note the thickness of the segment of bone (at lower part of the picture), which is also dense and ivory-like. (*Pathological Museum, McGill University.*)

three of them stand out pre-eminently. These are the thyroid, the pituitary, and the testes. The relationships that exist between these organs are numerous and cannot be entered into fully here. There can be no question, however, that a certain mutual balance of functional activity on the part of these structures is essential for the maintenance of normal growth and development. Should this balance be upset, metabolism is disordered and disease is the result. The body as a whole may be involved or some part of it. The changes are manifested

mainly in the direction of aplasia, hypoplasia, atrophy, or hypertrophy. A familiar instance of imperfect growth and development of this type is cretinism, which is now generally admitted to be the result of athyroidea. Of the opposite condition, namely, excess of growth, we may cite the elongation of the bones which occurs after castration. The posterior pair of limbs is usually increased in length in eunuchs, oxen, and capons. The overgrowth of hair that sometimes occurs on the faces of women who have passed the menopause, or who are the subjects of ovarian disease, is possibly also of this nature.

The most striking example, however, is *acromegaly*. In this curious disease, as Marie put it, there is a massive hypertrophy of bones of the extremities and of the extremities of the bones. The hands are spadelike and the fingers rounded. The lips, tongue, nose, cheeks, and ears become thickened, and a characteristic prognathic facies is in time produced. In severe cases all the bones of the body are affected. The thorax enlarges and the trunk becomes scoliotic. With this there are minor peculiarities, general physical and mental asthenia, trophic disturbances of the skin, and sexual apathy. In the vast majority of cases some lesion of the pituitary body has been found, such as hypertrophy, cystic or adenomatous tumors. Whether, however, the disease is the result of an increased, a diminished, or a perverted pituitary secretion is a question which at present must be unanswered.

Lastly, we have to discuss certain hypertrophies, which, for want of a better explanation, may be called neurotrophic. Such are, possibly, the cases known as "idiopathic hypertrophy of the heart." Here, the heart is hypertrophied in the absence of all of the ordinary conditions which produce this, such as arteriosclerosis, nephritis, emphysema of the lungs, and valvular lesions of the heart. Cases have been attributed to mental overwork, worry, and the abuse of tea and coffee. It is conceivable that in such cases there may be some functional disturbance of the nerve terminals or ganglia in the heart muscle. Of a similar nature appear to be those cases of hypertrophied bladder which are occasionally seen in children who suffer from frequent and painful micturition, with most of the symptoms of calculus. No calculus is present, however, and at autopsy no disease of the urinary organs is found other than hypertrophy. The condition appears to be due to a too frequent and powerful action of the vesical muscle. Possibly it is to be attributed to a spasmodic contraction of the muscles about the urethra, causing a temporary obstruction. This incoördinate action of the muscle is very likely the result of disturbed innervation.

ATROPHY.

The term "atrophy," from its derivation, implies wasting, lack of nourishment. In pathological language it means a condition or process in which the cardinal feature is diminution in size of a tissue or organ, either from a decrease in

the size of its constituent cells or from a diminution in the number of these cells, or both. We may, therefore, distinguish between a *quantitative* and a *numerical* atrophy. The distinction is, however, entirely theoretical, for it is practically impossible to separate the two conditions. Provided that the cause remain constantly acting, a cell which at first only becomes diminutive will in time disappear altogether. Atrophy is in most respects the antithesis of hypertrophy. In hypertrophy the size and number of the tissue elements increase, resulting in augmentation of function. The affected part becomes larger. In atrophy the part wastes from diminution of its substance, and its function is correspondingly impaired.

As we have seen in the preceding section, we have two kinds of hypertrophy—hypertrophy with *growth* and hypertrophy with *development*. Similarly we may recognize two forms of atrophy—atrophy with simple *wasting* and atrophy with *degeneration*. It is not always possible to make this distinction in any given case, for, as a matter of fact, a wasting tissue not infrequently degenerates, and, conversely, a degenerated tissue is usually smaller. Still, however, it conduces to precision of thought to preserve this distinction in our minds and to use the term "atrophy" in a more restricted sense, namely, to indicate a simple loss of substance, without connoting any other retrogressive change. It is well, also, before going further, to get a clear idea of certain terms which are not infrequently confused with "atrophy." These are agenesia, aplasia, and hypoplasia. *Agenesia* and *aplasia* mean complete failure of a part to develop. It is rare, however, for this to be absolute. *Hypoplasia* is underdevelopment. The causes which induce these peculiarities operate at different periods of life. Aplasia and agenesia arise during early fœtal existence; hypoplasia occurs somewhat later, but before complete development has been attained. All blighting or imperfect development of parts is to be regarded as aplasia or hypoplasia. Atrophy may occur at any time during the life of the individual and implies a retrograde decrease in size after the affected part has been developed, either completely or as far as it will go. The same causes which may induce atrophy in a perfected organ may, on occasion, produce it in one imperfectly developed, so that we must broaden our definition of atrophy to include all cases of diminution in size of the cells of a part, whether these cells be perfect or imperfect. Degenerative atrophy differs from simple atrophy in that the retrogressive changes which are present begin in the cytoplasm and nuclei, and later give rise to the decrease in size.

We can understand the philosophy of atrophy only if we constantly bear in mind the principles already enunciated. As we have seen, all cells possess an inherent vital energy, which is manifested in the functions of assimilation, nutrition, and reproduction. Continuance in life, to say nothing of growth and development, depends solely upon the maintenance of a certain balance between the nutritive or building-up forces and the destructive or wasting processes.

During early life, when anabolism is in excess, growth and development and the manifestations of a vigorous energy are dominant features. Later, there comes a period of equilibrium, during which the bodily powers are at their highest consummation. Later still, when the natural decay sets in, the faculties begin to fail and the machinery to give out, until the various functions can no longer be performed and the individual dies. Nutrition and waste are the two opposing powers. Atrophy of tissue may, on the one hand, result from imperfect nutrition and diminished repair, and, on the other, from excessive consumption and waste. In the first instance nutrition of tissue is largely dependent, apart from the inherent vegetative force before referred to, on a sufficient supply of healthy blood and lymph, and on the existence of proper nervous stimuli. Any disease process which interferes with the amount of blood reaching a part, or deteriorates its quality, or, again, cuts off the cells from their neurotrophic centres, results in atrophy. On the other hand, anything which increases tissue waste, as, for instance, overactivity, will give rise to atrophy.

It should be remarked here that there is a general law in pathology which governs the extent and the localization of retrogressive processes, atrophies included. The more delicate and highly specialized a cell or tissue is, the more susceptible it is to external impressions; and, when injured, the less its power of repair. Therefore, the parenchyma of an organ, the secreting structure of a gland, suffers more from deteriorating influences than does the stroma. The epithelium of the kidney and the parenchymatous cells of the liver may waste without material diminution in the connective-tissue framework. As a consequence, such an organ, when the subject of atrophy, often becomes harder and more fibrous than normal, or the destroyed cells are replaced by newly formed connective tissue. This change is commonly called *induration*. In such a case the organ affected becomes not only smaller, but its surface is often irregular, nodular, or warty. A change in external size and configuration is, however, not necessary in all cases. For example, in bone, atrophy may affect the trabeculæ of the spongy portion and the parts bordering on the medullary cavity. The bone in this way becomes lighter and more porous, but may not be smaller (*osteoporosis*).

Microscopically, the cells of the affected organ are smaller and usually fewer than normal. In the early stages of the process they usually stain well and the finer structure is well preserved. In more advanced cases the cells may be considerably shrunken and deformed, and there may be either an absolute or a relative increase in the pigment (*atrophia pigmentosa*). Pigmentary changes are often well seen in the case of the heart muscle and the secreting cells of the liver. Atrophied ganglion cells are generally highly pigmented. In the most advanced stages the cytoplasm has all but disappeared and little remains but a shrunken, distorted nucleus, which in its turn fragments and disappears. In certain cases, as in muscle, the loss of substance is made good, so far at least as bulk is concerned, by an overgrowth of the connective tissue in which fat is eventually

deposited (*atrophia lipomatosa*). This appears to be an attempt at compensation.

It may be inferred from what has already been said that we have to recognize two broad classes of atrophies—those which are *physiological* and *inevitable,* and those which are *pathological* and *accidental.*

Physiological or histogenetic atrophy results from the diminution of the inherent vital powers of the cells. It implies diminished repair rather than excessive waste. The potential energy of each cell and tissue is directly proportional to the amount of work it has to do. As the functional importance of an organ diminishes, so does that organ begin to atrophy. When any organ becomes useless in the economy it quickly disappears. Each organ and tissue has its own life period, and at the termination of its career inevitably dies, even should it have at no time been attacked by disease. The phenomena of atrophy are to be observed in the individual from the earliest embryonic life period. Numerous examples of this truth can be cited. In the formation of the placenta certain parts of the membranes disappear at an early stage of development, and with the formation of the chorion there is coincidently a progressive atrophy of the villi. The full-term placenta has fulfilled its purpose and is therefore cast off. In the case of the fœtus itself, the Wolffian and Muellerian ducts, the Wolffian bodies, the umbilical vesicle, and the omphalomesenteric duct disappear quite early. Before birth certain blood-vessels begin to be obliterated, and a few days after birth the closure of the Ductus Botalli and the umbilical vessels has already taken place. The spontaneous separation of the umbilical cord is also a manifestation of atrophy. Later the milk teeth are cast off. After puberty the thymus gland, which at first is one of the most prominent structures in the body, rapidly wastes away. With the approach of middle life, lymphoid structures begin to waste and portions of the petrous and sphenoid bones disappear. Hyaline cartilage in some persons is in time converted into bone. With the onset of old age the uterus and ovaries begin to shrink, and with the induction of the menopause their function ceases. This atrophy of the ovaries is due to sclerotic changes in the ovarian arteries, in which involution changes seem to begin sooner than in the vessels elsewhere in the body. In advanced life the lymphadenoid structures, the muscles, bones, and in many cases the subcutaneous fat, begin to retrograde. The lungs, kidneys, and liver are often considerably involved in the process, the brain and nervous system to a comparatively slight extent, as a rule. The posture, gait, the blanched hair, and general appearance of the aged are due to these atrophic changes. The cardiovascular system, on the contrary, may suffer slightly, if at all. The heart often increases in size and strength into advanced life, the blood corpuscles are formed as before, and defects of vessels, connective tissue, and epithelium are usually quickly made good. The arteries themselves, however, may undergo sclerotic changes. This is an important factor in the causation of the senile atrophy, in-

asmuch as the nutrition of the tissues is interfered with and in this way the normal retrograde processes are accelerated. In the category of physiological atrophy must also be included the normal involution of the uterus which ordinarily occurs after parturition. Some of these physiological atrophies are of considerable practical importance. The brittleness of the bones in old age is an important predisposing cause of fractures. Fracture of the neck of the femur is not uncommon and may result from but slight violence. Not only the osseous fragility is here to be taken into account, but also the alteration of the angle which the neck of the femur makes with the shaft, as it weakens the resisting power of the part.

Pathological atrophy may result (1) from inactivity or disuse, (2) from overactivity, (3) from impaired nutrition, (4) from pressure, (5) from neurotrophic disturbance.

Atrophy from Inactivity or Disuse.—The proper tone of a muscle and its functional perfection depend, on the one hand, on the amount and the kind of nutrition which it receives, and, on the other, on the regular and sufficient exercise to which it is subjected. A fully exercised muscle always has a good blood supply. A functionless muscle receives less nourishment than a normally acting one. The same principle may be applied to glands and other structures.

The loss of necessity for a function, or the inability to perform a function, whether from some incapacity of the organ itself or from any extraneous condition which interferes with the performance of its duty, may result in atrophy. The importance of the lack of proper function is seen in the case of certain deep-sea fishes, which possess only rudimentary eyes, in some cases covered with skin. The rudimentary, or, more correctly speaking, the remnant of the hind legs found in certain whales and reptiles is also a case in point. Again, in cases of marasmus or malassimilation the heart may be found to be quite small, below the average weight. This, in part at least, no doubt may be traced to the lessened demand upon its services. There is less blood, and therefore less force is needed to propel it. There is less demand also for oxygenation. In such cases the atrophy is to be attributed as well to lack of nourishment of the heart muscle itself. The atrophy of the acetabulum which occurs in cases of unreduced dislocation of the femur, the removal of the callus when a fractured bone has reunited, and the atrophy of the nerves after the amputation of a limb also come under this category. An organ or structure may be unable to perform its usual function, owing to some disturbance of its innervation or from some mechanical hinderance. Thus in paralytic cases the muscles and bones will in time waste. A similar result follows the immobilization of a limb, a deformity, fixation of a joint, or the presence of a tumor which interferes with the function of the part.

Atrophy from Overactivity.—It has been shown above that an increased demand upon the functional activity of a part results in hypertrophy. This process, however, has its limits. The moment the reserve power of the structure is encroached upon, we see the beginning of the end. The part gradually fails

to respond, and hypertrophy gives place to atrophy. Overuse of an organ acts by increasing waste and by giving the structure no time to recuperate. Therefore, elimination is diminished and the toxic products of metabolism accumulate in the tissues. Fatigue is the keynote of the process. The failure of compensation in a heart which previously may have been hypertrophied is a case in point. The brain is the chief organ which may be thus affected. Of glandular organs, probably the testicles most often suffer.

Atrophy from Impaired Nutrition.—This is the atrophy which is found typically in cases of starvation and in all forms of chronic wasting disease. It is sometimes called *marantic atrophy*. The condition depends in the main on the fact that the cells are receiving an insufficient amount of nutrition for their needs. The extent and the rapidity of the atrophy depend upon the degree of metabolic change which the affected part is able to undergo. Adipose tissue, for example, quickly disappears if there be any lack in the fat or fat-forming substances which are supplied to it. The bones become soft and wasted if the lime salts are withheld. It is likely also that the deficient amount of hæmoglobin in the red corpuscles is due to the deficient absorption of iron. Marantic atrophy may be general or local. General atrophy of the body is found in cases of starvation, whether from insufficient supply of food or from any condition of the digestive apparatus which prevents its proper absorption and assimilation. In such cases the fat, muscles, blood, and abdominal organs are chiefly affected. The fat disappears first, so that the angles of the body are exaggerated, the eyes sink in, and the whole body assumes a gaunt appearance. The muscles may be reduced to half their original size. The liver, spleen, and intestines, of the abdominal viscera, suffer most. The central nervous system, the bones, and the heart, on the other hand, show but little change. Curiously enough, lipomata do not decrease in size, even in cases where the ordinary fat of the body has disappeared.

Local atrophies are usually due to local causes. Thus disease or injury of blood-vessels may cause atrophy of the part supplied by cutting off the nourishment. Sclerosis or other conditions leading to obstruction or obliteration of vessels will produce atrophy of the part involved, usually with other degenerative changes. We find this, for example, in the heart, from obstruction of the coronaries; in the kidney, from obliteration of a branch of the renal artery; and in the brain, from a similar condition. Circulating toxins, by their deteriorating action on the blood and possibly by a direct local effect, may bring about atrophy. The long-continued use of iodine is sometimes followed by wasting of the thyroid and mammæ. In certain cases of fracture of the femur or of other bones, the circulation through the great nutrient artery may be cut off, and atrophy of the portion of the bone thus deprived of its food supply follows. Necrosis does not occur, because there are sufficient anastomoses remaining to preserve vitality.

Atrophy from Pressure.—It was pointed out above that pressure, if intermittent, especially if combined with friction, leads to hypertrophy. Continuous pressure, on the other hand, will produce atrophy. This is in part the result of direct injury to the cells at the site of the lesion, but it is also due to the circulatory disturbances induced. This form of atrophy, therefore, is passive in its nature. It is usually the result of slight pressure exerted over a prolonged period of time. Numerous examples might be cited. Among the best known are the "lacing" or "corset" liver and spleen, the foot of the Chinese lady, the flat head and flat

FIG. 61.—Atrophy of the Bodies of the Vertebræ from the Pressure of an Aneurism. (*Pathological Museum, McGill University.*)

nose of certain tribes of Indians. The pressure of tumors, aneurisms, cysts, varicose veins, may lead to atrophy of the adjacent structures (see Fig. 61). Malposition of bones, as in scoliosis, genu valgum, pes valgus, by altering the direction of the pressure, will lead to atrophy of the parts unduly pressed upon. The calvarium may be thinned from the pressure of a hydrocephalic brain, from tumors, and from enlarged Pacchionian bodies, or, externally, from wens. Disappearance of the alveolar processes of the jaws sometimes results from the loss of teeth, the bone being in this case subjected to unusual pressure. In congenital dislocation of the hip-joint, reposition of the head of the femur, with

maintenance in the new position, will lead to the excavation of a new acetabular cavity.

Perhaps less conspicuous, but equally complete in their way, are the local atrophies which result from the pressure of inflammatory exudates, scars, and constricting fibrous bands. In the so-called "nutmeg" liver the columns of parenchymatous cells about the centrilobular vein present extensive atrophy, usually combined with fatty degeneration, the result of pressure from the overdistended capillaries. Nutritional defects also, no doubt, play a part.

Neurotrophic Atrophy.—It is generally held that there are certain nerve cells or ganglia which, to some extent at least, preside over the nutrition of the tissues and structures innervated from them. These trophic centres are for the most part situated in the anterior horns of the spinal cord. Any lesion which destroys these cells or interferes with the conductivity of the fibres in the lower motor neurones is followed by wasting of the parts with which they are connected. Anterior poliomyelitis, progressive muscular atrophy, bulbar paralysis, are examples of this. In syringomyelia and tabes dorsalis atrophy of the bones and joints may occur. The so-called Charcot's joints are essentially atrophic in their nature. Disorders of the peripheral nerves may be followed by such manifestations as glossiness and atrophy of the skin, exfoliation, loss of hair, and disappearance of the cutaneous glands. The diminution in size of one-half of the body which results from unilateral disease of the brain in fœtal life and early childhood, while usually designated hemiatrophy, is probably more correctly to be regarded as a unilateral hypoplasia. Many of these forms of atrophy resulting from organic nervous disease are of practical importance to the surgeon, inasmuch as serious deformities of the members may result. This comes about from the overaction of certain muscles which are unopposed by the wasted muscles. Troublesome contractures not infrequently occur. Bedsores, especially those which develop rapidly in cases of organic nervous disease, are in large part trophic in character.

It should be remarked that, in the class of cases just referred to, neurotrophic disturbance is not the only factor to be taken into account. The loss of nervous impulses leads to paralysis of the muscles, and this brings on the atrophy of disuse. Again, certain vasomotor changes are induced, which interfere with the nutrition of the part. As a result of these alterations inflammatory processes are readily set up, induced by causes which would otherwise be ineffectual. Probably the neurotrophic atrophies are examples of degenerative rather than of simple atrophy.

Atrophy as It Affects Certain Tissues and Organs.—*Muscle.*—Voluntary muscles when atrophic may be much reduced in size, owing to thinning and disappearance of the individual elements. The muscles are also paler and softer than normal, dry and anæmic. They are frequently tough, owing to a relative or, in some cases, an absolute increase in the interstitial connective tissue. In

such cases the muscle as a whole is somewhat grayish in color. The amount of wasting may be so extreme as to give the impression of there being nothing between the skin and the bone (*living skeleton*). In other cases, owing to an increased deposit of fat between the fibres, the muscle appears to be of normal size (*atrophia musculorum lipomatosa*), or it may be even larger than normal (*pseudo-hypertrophic muscular paralysis*). The natural pigment of the muscle fibre may be relatively or absolutely increased, causing the muscle to assume a brownish appearance. This is often well seen in the heart (*brown atrophy*). Microscopically, the individual fibres appear to be shrunken, and are often tortuous. The striæ are usually well preserved. The nuclei of the endomysium proliferate more or less. When the atrophy is complete the sarcolemma sheaths are found to contain pigment, nuclei, and multinucleated cells.

There are one or two important forms of *muscular* atrophy which deserve more than a passing mention.

Atrophy from disuse may be brought about by fracture of a muscle, tendon, or bone, ankylosis of joints, fixation by splints, or even by voluntary inactivity.

In the neuropathic atrophies the process is largely confined to certain muscles or groups of muscles. These atrophies may be spinal, bulbar, or cerebral in origin, and often attack the muscles that are most used or those which derive their innervation from some diseased portion of the central nervous system. In manual laborers the muscles of the thenar and hypothenar eminences, the interossei, and the lumbricales are apt to be first involved (Aran-Duchenne type). In other cases the disease begins in the muscles of the shoulder and arm.

In cases of involvement of the medulla there is difficulty in articulation and deglutition, with drooling of the saliva and feebleness of the voice (*progressive bulbar paralysis*).

Somewhat closely resembling the spinal atrophies is the so-called *primary myopathy* or *progressive muscular dystrophy*. There are three types—the infantile, the juvenile, and the adult. The first usually begins in the muscles of the face, giving rise to a peculiar, expressionless appearance, the so-called myopathic facies. The juvenile form involves the muscles of the calves, thighs, back, shoulder girdle, and arms. The adult type begins either in the lower extremities or in the upper extremities and face. The anatomical cause is still a matter of dispute. The pseudo-hypertrophic form of primary myopathy occurs in children, and affects the muscles of the calves or shoulders, giving the child the appearance of a young prize-fighter. The myopathies are distinctly of a family type. They have a practical bearing for the surgeon, in that, when affecting the lower extremities, they may produce club-foot.

Secondary atrophies of muscles may occur when nerves are cut in the course of operations.

The heart, when atrophied, is reduced in size, the color is darker than normal, and the coronary vessels are tortuous. The epicardial fat is likewise wasted and

in a serous condition. Atrophy of certain portions of the heart may result from coronary disease or fatty infiltration. Atrophy of certain fibres is often combined with hypertrophy of others.

Lungs.—In the so-called hypertrophic emphysema there are atrophy and secondary rupture of the alveolar walls, so that the spaces become enlarged. Such

Fig. 62.—Caries of the Upper End of the Femur: Abscess in the Great Trochanter. (*Pathological Museum, McGill University.*)

lungs are more bulky than normal, pale, and anæmic. In another form, atrophic emphysema, the same condition is combined with a diminution in bulk of the whole lung.

Bones.—Atrophy of bone may result from any of the causes hitherto set forth. Old age, pressure, interference with function, impoverished nutrition, inflammation, and nervous disturbances may all play a rôle.

The structure of the bony framework of the body is in health undergoing constant change. In the child vegetative forces are in the ascendant, with the result that any loss of substance of the bone is made good and more than made good, so that the bones increase in size and strength. In the adult, breaking

down or resorption is still going on, but is compensated by a continuous deposit of new bone through the process of apposition. In the aged, resorption is in excess, and therefore the bones become lighter, smaller, and more fragile. Lacunar resorption is the process by which atrophy takes place. Certain large, multinucleated cells—osteoclasts—are present in great numbers in the periosteum and bone marrow. These take up their position on the bone trabeculæ and gradually erode the bone, forming the so-called Howship's lacunæ. In the rapid resorption of bone characteristic of certain diseases the osteoclasts are greatly increased in numbers and lie closely packed together. The result is that the surface of the bone becomes eroded and irregular (*concentric atrophy*) (see Fig. 62). Should the process go on mainly about the medullary cavity, the external configuration of the bone is not altered, but the marrow space is enlarged and the bone becomes thinner (*excentric atrophy*). In still other cases the compact portion of the bone becomes porous, owing to the widening of the Haversian canals (*osteoporosis*) (see Fig. 63).

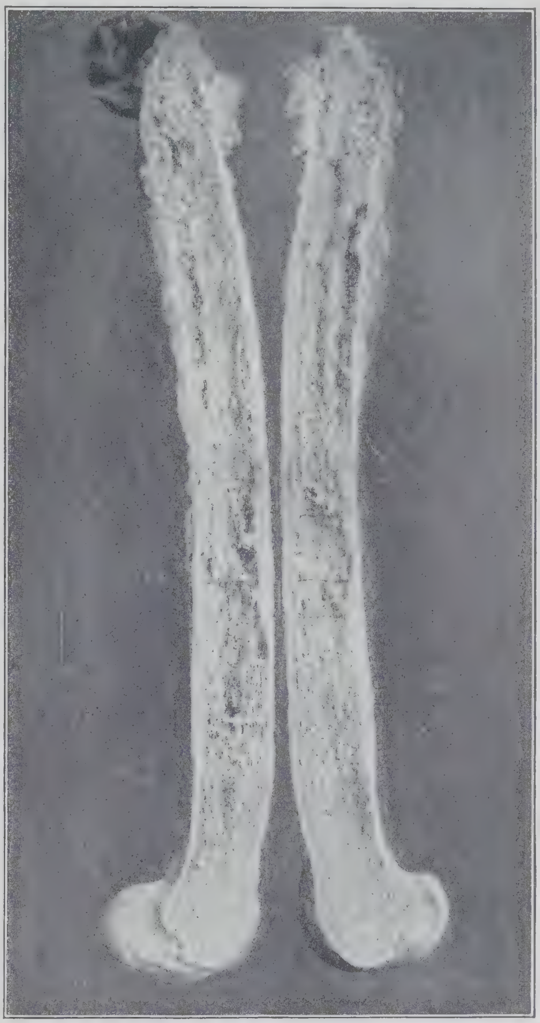

Fig. 63.—Femur Cut Longitudinally, to show Rarefaction, Osteosclerosis, and New Growth of Bone from the Periosteum. Case of Osteomyelitis. (*Pathological Museum, McGill University.*)

Atrophied bones are light, fragile, easily broken or sawn. The medullary substance is often lymphoid in character, fatty, or the fat may be replaced by a gelatinous-looking material (serous atrophy).

Senile and marantic atrophy may affect the skeleton as a whole, but the senile form is apt to attack more extensively the flat bones, the calvarium, maxillæ, scapulæ, and pelvis. The process begins and is most marked at the points devoid of muscular attachment. Owing to the diminution in size of the vertebræ and the intervertebral discs, the height of the body is diminished and the back becomes curved. The character-

istic senile facies is due to the absorption of the alveolar processes. Atrophy from disuse is more usually found in the limbs.

The atrophy that affects the bones after amputation is from without and leads to the end of the bone becoming thinner and more pointed.

Striking instances of atrophy from pressure are met with in cases of carcinoma and sarcoma developing within a bone. Not only is the bone rarefied but the structure is expanded. This is the cause of the so-called "egg-shell crackle," which is one of the clinical features looked for (see Fig. 64).

Fig. 64.—Humerus; Carcinoma of Upper Portion, showing Rarefaction of the Bone. (*Pathological Museum, McGill University.*)

From the point of view of structure the bones may be regarded as consisting of a living matrix, in which is deposited an inert, dead material, the lime salts, which to a large extent give compactness, density, and strength to the tissue. The relative proportions between these two elements may be considerably altered, a state of things which may possibly with some reason be considered as a form of atrophy. The proportion of calcareous salts is increased in old age, an expression of what one might appropriately call the "calcareous diathesis," which is so characteristic of old age, being found not only in the bones, but in the cartilages and blood-vessels. This does not result in an increase of strength of the bones, but, on the contrary, they become more fragile than normal, owing to excessive resorption.

An opposite condition to this is *halisteresis* or *mollities ossium,* in which the organic constituents of the bone remain unaltered, but there is a notable diminution in the amount of lime salts, so that the bone becomes soft and yielding.

The most extreme form of this is known as *osteomalacia.* The pathological changes at work here consist, in the main, of decalcification of the old bone, with

at the same time a tendency to the formation of new bone, which, however, remains imperfectly calcified. The process of decalcification begins at the periphery of the bone trabeculæ and gradually extends to the deeper parts. The line of demarcation between the normal and the diseased bone is sometimes even and continuous, but may be irregular and with excavations like Howship's lacunæ. Often there is formed an intermediate zone, in which the lime salts are not completely removed, but remain in the form of a crumbling detritus. Eventually the bone canals become enlarged, and, with the absorption of the calcareous salts, new canals are formed in the ground substance. The matrix itself may be homogeneous or may present a finer or coarser fibrillation. Some of the bone corpuscles may be preserved, but many have atrophied or disappeared, leaving small cavities. In some cases there is a new formation of osteoid tissue, but it remains for a long time, or even permanently, uncalcified. This new tissue may be quite dense, containing only a few spaces, or it may present a laminated or fibrillated structure with large corpuscles. Osteoclasts and Howship's lacunæ are not more numerous than in normal bone. The condition of the marrow varies. It may be reddish in color, with giant cells, yellowish and fatty, gelatinous, or even fibroid. Hemorrhages and pigment are commonly to be found.

Osteomalacia is a disease of obscure etiology. It appears to be rare on the North American continent, Dock finding records of only ten cases. Since some cases in women have been cured by removal of the ovaries, Fehling has promulgated the view that it is a trophoneurosis due to reflex irritation from the ovaries.

A condition of some practical interest to the surgeon is *osteopsathyrosis*, or abnormal brittleness of the bones. This term is used in a general way as synonymous with *fragilitas ossium*. The bones become fragile, and are therefore easily broken. A slight movement, a jar, or even muscular action may be sufficient to bring about this result. The condition is a retrograde metamorphosis, and is met with especially in senile and cachectic conditions. Rarely, the condition is congenital and may be inherited. In this case it appears to be a developmental anomaly, for it has been found combined with dwarfism or associated with dwarfism in related individuals.

Osteopsathyrosis occurs as a result of old age, cachexia, prolonged activity, pressure atrophy, and neurotrophic atrophy. It is met with in locomotor ataxia, syringomyelia, general paresis of the insane, and osteomalacia. It is also found in certain inflammatory conditions, such as rickets, syphilis, and leprosy. In inveterate syphilis with cachexia there is often a marked fragility of the long bones especially, and also of the cranium. A somewhat similar condition of osteoporosis is the result of actinomycosis and Madura foot disease.

The Nervous System.—The subject of atrophy of the nervous system is one that is beset with great difficulties and uncertainties. Here, as in so many cases, we cannot draw any distinction between atrophy and degeneration. We may

be certain, however, that they are always combined. The nervous tissue, above all structures, is particularly liable to undergo retrograde changes. It is the most delicate and highly specialized mechanism in the body. It is, therefore, especially susceptible to the deteriorating action of a variety of agencies, while its regenerative powers are but slight. Disintegration and degeneration are consequently the most frequent and important pathological changes to which nerve tissue is liable. It is, again, not always possible in any given case to determine the etiological factor chiefly or entirely to blame. Thus, the distinction between inflammatory and simple degeneration is often obscure.

In order to get a clear appreciation of the degenerations affecting the nervous system, it is important to bear in mind certain facts.

According to the "neurone concept" of the histological structure of the nervous system, most generally accepted at the present day, the brain and cord, with their continuations, the peripheral nerves, are to be regarded in general as a peculiar association of highly specialized cells, consisting of a large cell body with protoplasmic processes (the ganglion cell), from which proceeds a single, long, and attenuated thread (axis cylinder or neuraxone). These together constitute the neurone. Each neurone is, so to speak, self-contained, and has no association with adjacent neurones save by contiguity. The protoplasmic processes (dendrites) are richly branched and interlace freely about the cell body. They are believed to be nutritive in their function, and they convey impulses toward the cell body. The axis cylinders give off collaterals at different levels and terminate in a complicated arborization, usually about another cell, at a considerable distance from their point of origin. The impulses passing through them are centrifugal.

In general terms it may be stated that when a neuraxone is for any cause cut off from its nutrient centre, the ganglion, it will degenerate. The process begins at the distal extremity and gradually progresses backward to the site of the lesion. When the degeneration is confined to one physiological tract of the brain or cord, we speak of a "system" degeneration. When one or more such tracts are involved, we have a "combined system degeneration." Similar results will follow any cause which interferes with the conductivity of the nerve fibre. In what are often called "primary" degenerations, in which the nerve bundle is acted upon directly and locally, certain tracts appear to be specially picked out, namely, the sensory neurones of the cord; the central motor neurones, beginning in the pyramidal layer of the cerebral motor cortex, extending through the internal capsule into the pyramidal tracts; and the peripheral motor neurones, which begin in the ganglia of the ventral cornua, passing through the anterior roots to the muscles. In "secondary" degeneration, due to causes acting from a distance, we have *ascending* and *descending* forms, according to the direction which the degeneration takes in the cord. Ascending degeneration affects usually the posterior columns, the direct cerebellar tract, and the antero-

lateral tract of Gowers. Descending degeneration affects mainly the pyramidal tracts.

There are many causes of nerve degeneration. Chief among them may be mentioned old age, mechanical trauma, pressure, circulatory disturbances, and various intoxications. In many instances more than one factor is at work.

Simple atrophy of the brain is particularly well exemplified in old age. The organ as a whole is diminished in size, the weight being below the normal. The wasting is most marked in the frontal and vertical regions. To gross appearance the convolutions are small and the sulci wide. On section the cortical gray matter is seen to be somewhat thinned. In the more advanced cases, in addition to these changes, the perivascular lymph spaces are enlarged, so that the vessels lie in wide channels. Small foci of degeneration are often to be seen (*état criblé*). In such cases it is not uncommon to find enlargement of the subarachnoid space and ventricles, which may be filled with fluid (hydrops ex vacuo). The cerebellum, as a rule, escapes.

Histologically, the lesion is atrophy of the specific nerve elements, ganglia and medullated fibres.

Senile atrophy is to be attributed to several causes, among which may be mentioned the normal tendency to retrogression evinced by all tissues as advanced life is approached, impoverished nutrition of the body, and the local effects of a scanty blood supply, due to the sclerosis of the vessels which is so constant an accompaniment of old age. In accordance with the rule that the more highly developed structures are the most liable to disease, it is that portion of the brain which has to do with the intellectual functions which suffers most.

There are other atrophies, such as those local ones due to vascular changes, which might be mentioned, but they are more properly discussed elsewhere.

Among the commonest forms of degeneration of the spinal cord is that due to pressure, the so-called "compression myelitis," a condition of much practical interest to surgeons.

As a rule, the lesion is a transverse one, affecting all the tracts of the cord in a comparatively restricted area. The usual causes are fracture of the vertebræ, tuberculous caries of the spine, tuberculosis of the spinal meninges, tumors of the vertebral canal or of the cord itself. Central degeneration may be caused by an accumulation of blood or fluid in the central canal. As a result, we get marked interference with and, finally, destruction of the nervous structures at the site of the lesion, with widespread ascending and descending degeneration in the associated tracts. The local effects are to be referred in part to the direct influence of the pressure, but probably more to the disturbance of the blood and lymph circulation. In some cases inflammation is also an associated cause.

The degeneration is first manifested in the white substance, the fibres of which swell up and disintegrate. The neuraxones swell and become varicosed, and the myelin sheaths break down into fat. The ganglia are somewhat more

resistant. Granular cells appear early and in considerable numbers. In the course of a few days the degeneration reaches to the terminations of the neurones. Later, both the fibres and their sheaths almost entirely disappear, although degenerated and varicosed fibres may be found here and there, the number of fibres remaining being dependent on the extent and severity of the original cause. The place of the destroyed fibres is taken by newly formed glia, which in time leads to contraction and sclerosis of the cord. The cord as a whole shrinks, becomes firmer, and assumes a grayish color. In some cases, where the pressure can be relieved by operation, the destroyed fibres will to some extent be replaced and function may, in part at all events, be restored. The attempt must, however, be made early.

Among other affections which may possibly be put in this category of atrophy may be mentioned disseminated sclerosis, anterior poliomyelitis, progressive bulbar paralysis, progressive spinal muscular atrophy, lateral sclerosis, postero-lateral sclerosis, amyotrophic lateral sclerosis, tabes dorsalis, pellagra, and chronic ergotism. In how far these are due to simple atrophy and how far to inflammation, it is impossible to say. Defective nutrition, dependent on an impoverished blood supply, is a probable cause in some cases, as, for example, in the changes in the posterior and lateral columns of the cord in pernicious anæmia. The influence of toxins may be traced in other cases, as in ergotism, pellagra.

Many of these degenerations are important in their results, as they lead to muscular atrophy, contractures, and even malformation.

Atrophy of the peripheral nerves, as of the cord, is not to be dissociated from the idea of degeneration. It may result from any cause which interrupts the continuity of the fibres with their nutrient centres, or which destroys the trophic influence of the centres themselves. Destruction of the ganglia either in the brain or in the cord is followed by degeneration of the fibres proceeding from them. Severance of a nerve trunk, as in an accident or in the course of an operation, pressure exerted continuously upon a nerve trunk, and inflammation are common causes.

Degenerative atrophy of a nerve fibre usually begins at the point most remote from the trophic centre, and proceeds centripetally.

The degeneration which results from severance of a nerve trunk, known as Wallerian degeneration, may be taken as a type of all the rest.

When separation takes place by means of a clean cut, all the fibres degenerate practically simultaneously and, moreover, speedily. Within twenty-four hours after the injury the material composing the medullary sheaths becomes cloudy and less refractive. After about three days indentations and other indications of segmentation are to be seen in the medullary sheaths and sheaths of Schwann, which later break up into droplets of myelin and fat. Next, the axis cylinders show evidences of atrophy and gradually disappear. They swell up,

become varicosed, and vacuolated. The interstitial substance is little if at all affected. The complete removal of the products of the degeneration may be long delayed.

Eventually the whole of the nerve distal to the point of injury may be destroyed. Degeneration in a centripetal direction occurs, but is comparatively unimportant, usually stopping at or close to the nearest node of Ranvier. Where the injury has been severe, isolated fibres may degenerate somewhat farther, and slight retrogressive changes may occasionally be made out in the ganglia.

Somewhat similar, if indeed not identical, changes may be brought about by the action of circulating toxins and bacteria, impoverished nutrition, and certain circulatory disturbances. Systemic anæmia and marasmus are important in this connection. Endarteritis and obstructive conditions of the blood-vessels bring about atrophy, largely by cutting off nutrition.

Degeneration affecting one or more nerve trunks occasionally arises in the course of diphtheria, influenza, typhoid fever, typhus, smallpox, tuberculosis, the puerperium, and in chronic poisoning with mineral substances, such as lead. Here, in some cases at least, we have the combined effect of imperfect nutrition and the deleterious action of the toxin.

Occasionally, owing to some unknown infection or intoxication, the trophic ganglia in the anterior cornua of the spinal cord are destroyed—a condition quickly followed by degenerative changes in the fibres proceeding from them.

Secondary atrophy of a nerve trunk may result from inactivity of the muscles it supplies, whether from paralysis or from fixation of the part. The nerves of special sense do not escape this process. Thus the optic nerve and tract will atrophy in cases of blindness.

The Skin and Associated Structures.—Simple atrophy of the skin is manifested by a wasting of almost all the elements entering into its structure. It may be generalized or localized. It may, again, be primary or the result of some external pathological condition.

As a type, we may consider the physiological atrophy which takes place in old age. In this case the cutis is thinned, the papillæ are flattened and tend to disappear, while the epidermis becomes dry and brittle. Owing to the absorption of the subcutaneous fat, the skin is thrown into folds. The elastic tissue involutes and the superficial vessels are degenerated. Brownish pigment may be found in the cells of the rete and about the vessels of the cutis. The deeper portions of the epidermis are wasted, so that the stratum corneum is less widely separated from the papillary layer. The hair follicles participate in the process, the hairs lose their pigment, become downy, and finally fall out. Not infrequently the openings of the hair follicles become obstructed, owing to the accumulation of epidermal scales, so that the follicles may become dilated into cysts. In the same way the sebaceous glands may be involved, and hair follicle and gland may be expanded into a cavity containing hairs, fat, and epithelial

débris, the so-called atheroma or wen. Eventually the sebaceous glands may disappear. Not uncommonly the superficial epidermis is in places heaped up into scales (pityriasis simplex).

Local atrophy is a common condition brought about by distention of the skin from any cause. It is found usually on the abdomen, breasts, and thighs. The commonest cause is pregnancy, but tumors, lactation, ascites, and anasarca will produce it. During pregnancy the abdomen is covered with reddish, livid streaks, which after delivery are transformed into silvery lines or scars (lineæ albicantes). In such areas the papillæ are flattened or absent, the connective-tissue fibres of the corium are dissociated, and the elastic fibres and blood-vessels are atrophic.

Pressure, as from corsets or other clothing, may produce a similar effect.

Local atrophy of this type has been described as occurring in certain acute febrile processes, notably typhoid fever, and in chronic wasting disease where the subcutaneous fat has been absorbed.

An idiopathic, diffuse, symmetrical atrophy has been reported by several observers.

Lastly, atrophy of the skin may be neurotrophic in origin, and is met with in such conditions as lepra anæsthetica, neuralgia, and neuritis. The skin over certain areas supplied by the affected nerves becomes thin, smooth, and shiny, and there may be wasting of the glands and hair follicles.

Lymphatic Nodes.—Simple atrophy of the lymphatic nodes occurs as an involution process in old age. The same applies to diffuse lymphadenoid tissue wherever found. The lymphadenoid structures appear to be more active in children and young adults, and physiological retrogression occurs comparatively early.

The lymphoid cells are the structures chiefly affected, while the stroma is, on the contrary, relatively or even absolutely increased. The nodes are small, increased in consistency, and contain but little juice. Occasionally the lymphoid cells of the medullary portion disappear and the fibrous supporting framework undergoes fatty transformation. The process gradually spreads to the cortex, and the fundamental structure of the node may in time be entirely destroyed. This change is met with chiefly in cases of chronic alcoholism. The condition of the lymphatic nodes is of considerable importance in regard to the question of infection and inflammation, inasmuch as these structures have much to do in combating disease processes of this kind. As is well known, infective processes, especially inflammation and suppuration, tend to become localized at the points where the nodes are most in evidence. Should they be damaged, either from atrophy or from disease, they are no longer competent to perform their functions, and the dissemination of the infective process is rendered more easy. The normal involution process which lymphadenoid tissues undergo is, in a certain sense, conservative, since structures so affected do not take up bacteria to the same extent, and hence are not so liable to suffer. The involution of

the lymphoid tissue in the appendix is given as one of the reasons why appendicitis is less likely to occur after middle life than in the young and vigorous.

Spleen.—Atrophy of the spleen occurs in old age and in chronic wasting disease. It is practically of no importance. The organ is diminished in size, the capsule is wrinkled and opaque, and is thrown into folds. On section the substance is pale and the trabeculæ are prominent. The stroma may be absolutely or relatively increased, while the pulp is diminished.

Liver.—Simple atrophy is found in advanced age, in marasmus, and in the various cachexias. A common cause is pressure, as from corsets. This leads to the formation of deep furrows and eventually accessory lobes ("lacing lobes"). Carcinoma of the œsophagus and stomach is particularly apt to cause atrophy of the liver, probably from lack of nourishment.

The process affects chiefly the anterior edge of the liver. The parenchymatous cells waste, and eventually disappear until nothing is left but the supporting stroma. The edge of the liver thus becomes sharp and harder. In many cases the amount of pigment in the secreting cells appears to be increased (brown atrophy). Frerichs has described a special form of atrophy—melanæmic atrophy—said to be due to the blocking of the capillaries with black pigment. Acute yellow atrophy of the liver is presumably of an infective nature. It may be mistaken for the cholæmia of obstructive jaundice.

Stomach.—The part affected is the mucous membrane, which becomes thinned and the glands granular and diminished in size. Atrophy of the stomach occurs in old age, cachexia, and marasmus, and also as a result of chronic gastritis.

Testes.—Atrophy of the testes occurs in old age and as a result of wasting disease. It may also be caused by pressure, as in cases of hydrocele, hæmatocele, varicocele, hernia, and tumors. It is said to occur also as a result of injuries to the cerebellum, or of concussion of the brain, and in paraplegia.

The secreting cells of the tubules become fatty, waste, and disappear, while the connective-tissue stroma is relatively increased. A striking feature is the thickening of the walls of the tubules, which appear to be swollen, transparent, and hyaline.

Prostate.—Simple atrophy is met with in from twenty to thirty per cent of old men. Occasionally it is met with in the young, as a result of marasmus, cachexia, castration, the pressure of retained urine, pent-up secretion, or concretions, and in the impotence of tuberculosis.

In the form due to constitutional causes the glandular structure is the part chiefly affected, while in that due to concretions the stroma suffers most.

Ovaries.—Atrophy of the ovaries occurs as a senile change and as the result of chronic oöphoritis. The senile ovary is smaller than normal, firm, nodular, and of a grayish or pearly white appearance. The albuginea is hard and may be several millimetres thick. The follicles are largely converted into small fibrous

masses (corpora fibrosa), with marked thickening of the theca. The arteries are usually thickened and hyaline.

Mamma.—Simple atrophy of the glandular elements of the mammæ occurs as an involution process after the menopause, and occasionally, but by no means invariably, after removal of the ovaries. The wasting is often masked by an overgrowth of fat. Atrophy is also said to result from the prolonged administration of iodine or its compounds.

DEGENERATION.

Closely associated with atrophy, and in some instances not to be dissociated from it, are certain cellular changes which we call *degeneration*. The term "degeneration" is often used in a loose way to designate all kinds of retrograde metamorphoses, but it is also employed in a specific and more restricted sense to indicate a particular class of retrogressive phenomena, which are characterized by the formation of new substances out of the cell protoplasm. Such new material may be retained within the cells or discharged from them. An abnormal increase in the production of substances normally elaborated in the cell may also possibly be included in this category.

As we have seen in discussing the subject of growth and development, the vital force of cells is manifested in three ways, namely, in nutrition, in reproduction, and in function. These, again, are dependent upon metabolic processes in the cell, whereby the potential energy of the food is transformed into actual work. Disturbances of metabolism lead, on the one hand, to abnormal growth and development, and, on the other, to various forms of wasting and disintegration. As has been remarked above, we have carefully to distinguish, at least in our mental conceptions, between atrophy and degeneration, both of which belong to the great family of the retrograde metamorphoses. The former connotes a mere alteration in size, the latter a chemical synthesis of new substances. It may not perhaps be strictly correct to state that in atrophy there are no abnormal chemical processes going on, but at least they are in the background. In degeneration, chemical changes dominate the picture, and alteration in the size of a cell may or may not take place. Frequently, atrophy and degeneration are combined.

As in the case of atrophy and hypertrophy, we have to distinguish between physiological and pathological degenerations. To a certain extent degeneration is natural and not a manifestation of disease. The animal body, and, in fact, all living organisms, after a preliminary period of growth and development, reach a fastigium, and eventually pass into a state of decrepitude, which we call senility, or old age, characterized, so far as the cells are concerned, by inability to repair the normal waste, imperfect powers of proliferation, and diminished function. Each cell, each tissue, each complete organism has its own life period, and stead-

ily progresses toward inevitable death. This deterioration of substance and impairment of function are the concomitants of atrophy and degeneration, which may in many instances be regarded as way-stations on the road to the great terminus. The rate of this progress is, however, not uniform. Some tissues and structures become old and die sooner than others. The involution of certain organs may, as we have seen, occur during fœtal life, in childhood, or in early adult life. The process of involution is in part one of atrophy, and frequently one of degeneration as well. Physiological degeneration is the product of normal retrogressive changes. Senile involution may, however, take place prematurely. It is then pathological. Many of the degenerative manifestations are not expressions of a natural, though premature, tendency to involution inherent in the cells, but are the result of some deleterious influence acting from without.

Death of cells may be sudden or gradual. Rapid death of cells or tissues, without any previous abnormal changes in the cellular substance, is termed *necrosis*. Gradual death, preceded by diminution in size (atrophy), by abnormal intracellular chemical processes (degeneration), or by the abnormal deposition of foreign material derived from a distance (infiltration), is called *necrobiosis*. This distinction between sudden and gradual death is largely theoretical, and is only valuable in that it conduces to accuracy of thought and convenience of description, but it should be borne in mind that it is not always possible in practice to draw this distinction. While necrobiosis is invariably a sequel of atrophy or degeneration, the converse is not necessarily true, that pathological atrophy and degeneration inevitably lead to death of the cell or tissue. Provided that the cause is removed or is not constantly acting, the condition may be recovered from.

It is usual to classify the degenerations—this term being used in its narrower sense—according to the nature of the abnormal substances produced. Strictly speaking, true degeneration implies the manufacture of new chemical substances out of the protoplasm of the cells themselves. It is convenient, however, to discuss at the same time a somewhat similar process, that of *deposit* or *infiltration*, in which the abnormal substances are brought to the cells from some distant part of the body, or, it may be, are introduced from without, and are stored up as so much foreign material within the cell bodies. Such deposit may exist *per se* or be the result of degeneration in some other part of the body. As a matter of fact, the distinction between degeneration and infiltration is not always very clear, for the same or similar substances may be elaborated within the cell out of its own protoplasm or may be brought to it from afar. And, again, in the imperfect state of our knowledge, we are not always able to trace the exact course of events in any given case. It is generally held that some substances, such as fat, melanin, and glycogen, may be of the nature at one time of a degeneration, at another, of a deposit.

Warthin classifies the degenerations and infiltrations as follows:

True Degeneration.—1. Cloudy swelling. 2. Fatty degeneration. 3. Hy-

dropic degeneration. 4. Colloid degeneration. 5. Colloid-like bodies. 6. Mucin. 7. Pseudo-mucin. 8. Cholesterin. 9. Epithelial hyalin. 10. Cornification. 11. Pigments formed by cell activity. 12. Glycogen.

Deposits.—1. Fat. 2. Amyloid. 3. Hyalin. 4. Calcification. 5. Uric acid, urates. 6. Cholesterin, cystin, xanthin, etc. 7. Glycogen. 8. Pigment. 9. Extrinsic substances.

Cloudy Swelling.—Cloudy swelling (granular, albuminous, or parenchymatous degeneration) is the commonest of the true degenerations. Here the cytoplasm is broken up into fluid and granules of an albuminous material. The condition is found chiefly in the secreting cells of glands and in muscle, but is said to affect also, though to a less degree, the connective-tissue stroma and wandering cells. Organs so affected are somewhat swollen, pale, doughy, and less glistening than normal. In severe cases they have a parboiled appearance, which has been compared to raw fish.

Microscopically, the parenchymatous cells are, as the name would imply, swollen, cloudy, or opaque, and the nucleus may be indistinct or even invisible. The cloudiness is due to the presence of innumerable fine particles in the cytoplasm. These particles are so thickly placed that the normal structure and granulation of the cell body is obliterated. The particles are not fat, since they do not stain with osmic acid or Sudan III. They dissolve on the addition of weak acetic acid or caustic potash, inasmuch as they are converted into acid or alkali albumin, which is soluble. The condition is best seen in fresh specimens cut on the freezing microtome. In severe cases the chromatin of the nuclei breaks down and is diffused, and the cell may even disintegrate into a fine granular débris (see Fig. 65).

Cloudy swelling occurs in all infectious fevers, in various intoxications, and in cachexias. Thus it is often found in typhoid fever, variola, diphtheria, scarlatina, and septicæmia, in acute nephritis, and in poisoning from chemical substances, such as bichloride of mercury, carbolic acid, arsenic, and cantharides. The condition is, moreover, not to be regarded as the result of high temperature only, for it is met with in many afebrile conditions, such as carcinosis. Much more likely, it is to be attributed to the influence of some circulating toxin. The extent of the process is probably directly proportional to the amount of the circulating toxins.

The exact nature of cloudy swelling is not absolutely clear. Virchow, who first described it, found it in cases of parenchymatous inflammation, but it is undoubtedly often to be found in organs that are not inflamed. Nevertheless, it is true that in many instances it is the earliest manifestation of inflammation. There is also a close relationship between cloudy swelling and fatty degeneration, for the two are often combined, and, if the cause acts for a prolonged period, the former passes imperceptibly into the latter. If the condition be not extreme, recovery is possible, with complete *restitutio ad integrum.*

Fatty Degeneration.—It is generally taught that in this form of degeneration the fat is formed out of the albuminous material of the cell body. As we shall see, there are some grounds for doubting this. The structures usually affected are the parenchymatous organs, such as the liver and kidneys, but the muscles and connective tissue are by no means infrequently involved. All cells, moreover,

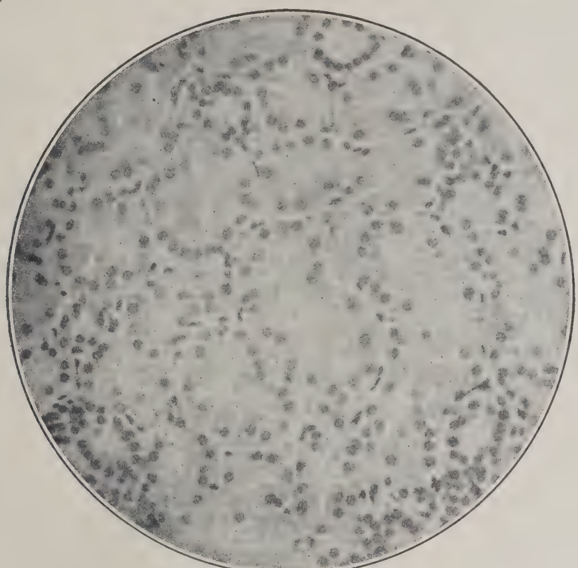

FIG. 65.—Cloudy Swelling of the Kidney. The secreting cells are swollen and cloudy; the nuclei stain badly; the lumina of the tubules are irregular. (*Leitz obj. No.* 7.) (*From the author's private collection.*)

which are out of their environment or are cut off from their source of nourishment, may undergo fatty degeneration, as, for example, leucocytes (pus cells) and carcinoma cells.

The affected organs are usually diminished in size, soft, friable, or doughy, and of a pale yellowish or yellowish-white color. On section the surface is often greasy. The condition may be uniformly distributed through the organ or may occur in specks, patches, or streaks. A well-known example of this is the so-called "thrush-breast" heart, found often in pernicious anæmia and in some febrile conditions. The affected organ is usually flabby and lacking in consistency.

Microscopically, the cell protoplasm contains numerous small, highly refractile, colorless droplets. These are very irregular in size and shape, in some cases being extremely minute, in others, where the smaller droplets have become confluent, forming larger drops, which may almost completely fill the cell. The droplets are insoluble in acetic acid, but are soluble in alcohol and ether. With osmic acid they take a brownish or blackish color (see Fig. 66). When treated with Sudan III the finer particles stain a golden yellow; the larger are more of a carmine color.

In general, fatty degeneration occurs under the same conditions as cloudy swelling. Given a sufficient intensity in the cause or a sufficient length of time, and cloudy swelling will pass into fatty degeneration. We find, then, fatty degeneration in acute infectious diseases, such as typhoid, pneumonia, diphtheria, scarlatina, septicæmia, erysipelas, etc., in poisoning with phosphorus, camphor, arsenic, alcohol, and chloroform. In regard to the last-mentioned agent, it may be remarked that several cases of acute fatty degeneration of the liver, followed by death, have been recently reported in connection with chloroform anæsthesia. This result is found in delicate or debilitated persons, especially those who have been suffering from chronic bone disease. Besides the causes mentioned, anæmia of all kinds, chronic congestion, diminished blood supply, may on occa-

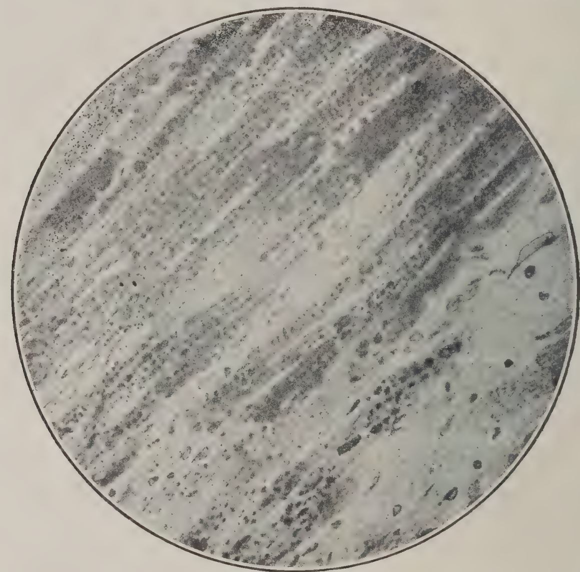

Fig. 66.—Fatty Degeneration of the Heart. Specimen stained with osmic acid. Fat is black. (*Leitz obj. No. 7.*) (*From the author's private collection.*)

sion produce it. Acute and chronic anæmia from hemorrhage, pernicious anæmia, leukæmia, and the local anæmia caused by sclerosis of vessels, embolism, or thrombosis, are important in this connection.

The conditions at the back of fatty degeneration appear to be impaired vitality of the cells, together with changes in the nutrition. The chief factor is believed to be deficient oxygenation of the cells. This results in the cell protoplasm being broken down partly into fat and partly into nitrogenous substances which are excreted by the urine.

Diminished oxygenation may be produced by a deficiency in the quantity of blood supplied to a part or by defective quality. By some, the albuminous and fatty changes so often found in febrile affections are regarded as due to the functional increase necessary to the production of the increased heat, especially in

regard to the organs which are most directly concerned in the maintenance of the bodily heat, viz., the heart and the liver. Probably, however, the direct influence of bacterial toxins and the toxic products of disturbed metabolism are of more importance. Possibly, too, increased functional activity, amounting to overstrain, may, as in the case of atrophy, lead to retrogressive changes of this character. This is rendered likely by the fact that fatty degeneration is often chiefly localized to organs which are apt to be overworked: the heart, from pumping an increased amount of blood—blood, moreover, which may be deteriorated in quality; and the liver and kidneys, which have to excrete the deleterious substances.

When we come to discuss the essential nature of the process resulting in fatty degeneration, we are led into somewhat uncertain paths. It has been almost universally taught by physiologists that fat is formed from proteid material. The pathologists have accepted this, and have assumed that in fatty degeneration the transformation is at the expense of the cellular substance. Taylor, Pflueger, Athanasiu, and others strenuously combat this view, holding that it has never been demonstrated to be possible from a chemical point of view. The question must in the mean time, perhaps, be left open. Bauer's experiments would, on the one hand, indicate the possibility of the older view. He found that he could produce extensive fatty degeneration in dogs by feeding them with phosphorus, in cases where they had previously lost their fats through a course of starvation. Lindemann and others also think that the marked cytoplasmic and nuclear changes in fatty degeneration are sufficient evidence of the origin of the fat from the cell protoplasm. On the other hand, even if it be true that fat is formed within degenerated cells, it is open to belief that it may possibly be formed out of carbohydrate substances present in the cells. It can hardly be denied, however, that cloudy swelling is due to a chemical transformation of the cellular proteid; and, inasmuch as cloudy swelling and fatty degeneration arise under identical conditions and are so frequently combined, the one condition often passing imperceptibly into the other, the proteid origin of the fat does not appear by any means unlikely. In view, however, of the unsettled state of the controversy, it would perhaps be more strictly correct to speak of "fatty degeneration" as "a cell degeneration associated with the formation of fat."

Fatty Infiltration.—Fatty infiltration may be defined as a deposit of fat in the cells of an organ or tissue, without any essential change in their structure.

Under ordinary circumstances fat is stored up in the cells in various parts of the body. It acts as a protection against external cold and injury, as a lubricant, and as a potential source of energy. The amount of fat present in any given case depends upon the quantity produced in the processes of assimilation and nutrition and the amount consumed in the oxidation incident to metabolism. Fat may be brought to the body in food, being emulsified and carried from the intestines by the lacteals and lymphatics and being stored up in the various cells of the body, notably those of the liver. Or, again, it may be pro-

duced by cellular activity from the carbohydrate and proteid constituents of the food. Therefore, an excessive amount of fat may accumulate in the system, if there be an abnormal amount ingested or produced, or if there be a deficient consumption of the fat. Or both factors may be at work. Up to a certain point the process of fat deposit is physiological, and we must be prepared to admit wide variations in its extent as being within normal limits. When it becomes excessive we speak of *obesity*, *adiposity*, *lipomatosis*, or *polysarcia*. The process, however, is in general the same, whether under physiological or under pathological conditions. What may be termed digestive fatty infiltration is well seen in the liver after a meal. On the other hand, when an animal is deprived of food for some time the liver becomes destitute of fat. An abnormal amount of fat may be laid down if there be an excess of fat in the food ingested. According to the older view, this fat was carried mechanically to the liver and deposited there. There are reasons, however, for thinking that the process is not so simple as this. The proportions of the various constituents of fat vary in the case of different animals. Now if you feed, for example, a dog on palmitin, this will be transformed and deposited in the various cells in the form of fat normally characteristic for the dog. This can be explained only by assuming the active interference of the cells themselves. Therefore, fatty infiltration is not a mere deposit of fat in passive receptacles, but a true metabolic process, in which the cells play a most important part. What happens is probably this: The fat ingested is immediately oxidized and employed for the requirements of the body; the fat that is deposited is the result of the metabolism of the carbohydrates and proteids brought to the cells. Whichever view is accepted, it is evident that fatty infiltration differs essentially from fatty degeneration. In the former the fat is not produced at the expense of the cell protoplasm, but from material imported to the cells from without.

Fat, again, may accumulate owing to deficient fat consumption. Here all conditions which lead to imperfect oxidation are important. Probably of this nature is the obesity which comes on with middle age. Diminished work on the part of the cells, sluggish respiration, chronic anæmia, alcoholism—all may play a part.

In some cases the normal balance which should exist between fat production and fat consumption is upset without obvious cause. The food is of suitable quantity and quality, and the waste appears to be going on normally, and yet, in spite of this, fat accumulates in excess. Here there are abnormal metabolic processes going on, the exact nature of which we do not understand. Undoubtedly in some instances there is some inherited peculiarity. In others the tendency is acquired. It is interesting to note in this connection the relationship which exists between the sexual system and fat production. For example, in young girls fat tends normally to be deposited in certain situations with the onset of puberty. Women not uncommonly become stout after the menopause or

on removal of the ovaries. Eunuchs are also often obese. Conversely, young women who become inordinately fat are frequently sterile.

Fatty infiltration may be local or general. Local lipomatosis is to a certain extent to be regarded as complementary in certain cases. Thus, the fat is often increased about atrophic or wasted organs. For example, the fat is increased about a contracted kidney, in the interstitial substance of the atrophied heart, and between the bundles of atrophic muscle. Multiple, circumscribed, and often symmetrically disposed, fatty tumors—*lipomata*—are probably to be ascribed to abnormal cellular activity. Adiposis dolorosa (Dercum) also comes under this category.

General obesity is not uncommon. Here the excessive deposit of fat takes place first at the points where fat is normally stored, namely, in the subcutaneous and subserous connective tissue, in the liver, in the bone-marrow, and, later, in unusual situations, such as in the wall of the heart, in the interstitial substance of the voluntary muscles, and in the submucous connective tissue.

The gross appearance of a fattily infiltrated organ is characteristic. In the case of the heart, the organ is enlarged, mainly from a great deposit of fat in the epicardium. On cutting through the wall, this fat can be traced in the form of pads and streaks between the muscle bundles. In places, especially near the apex, there may be but little muscle left.

In fatty infiltration of the liver, the organ is enlarged, unlike what occurs in fatty degeneration. The edges are rounded, and the tissue is doughy, pitting on pressure. In color it is yellow or yellowish-white. On section it is soft, friable, and greasy. Globules of fat can be scraped off with the knife. In advanced fatty infiltration the liver may actually float when placed in water. These examples illustrate, moreover, the two types of fatty infiltration. In the former the fat is deposited in the interstitial connective tissue; in the latter, within the parenchymatous cells.

Microscopically, the heart will show large masses of adipose tissue lying between the muscle bundles (see Fig. 67). In the liver the parenchymatous cells show no structural changes other than those due to the mere presence of the fat. In the early stages the cells contain small droplets of fat, which later coalesce to form larger globules, almost or quite filling up the cell. In this way we get a large oil globule, surrounded by a thin shell of protoplasm, the nucleus of the cell being crowded to one side, which gives the cell a characteristic signet-ring appearance. Where fat globules are present within the specific cells of an organ, it is not always easy to decide whether we have to do with fatty degeneration or with fatty infiltration. In the former we can, on careful study, usually make out degenerative changes in the cytoplasm and in the nucleus. The cells are also usually atrophic, while in fatty infiltration the cells are larger than normal.

Fatty infiltration, like fatty degeneration, may result in serious interference

with function. General obesity leads to inhibition of movement, sluggish respiration, weak heart action, and to some extent it slows metabolism generally. Fatty infiltration of the heart in time produces muscular insufficiency and occasionally rupture of the wall. The liver, on the other hand, is able to perform its functions comparatively well, even in the presence of advanced fatty deposit. These results are brought about in part by the mechanical effect of the deposited

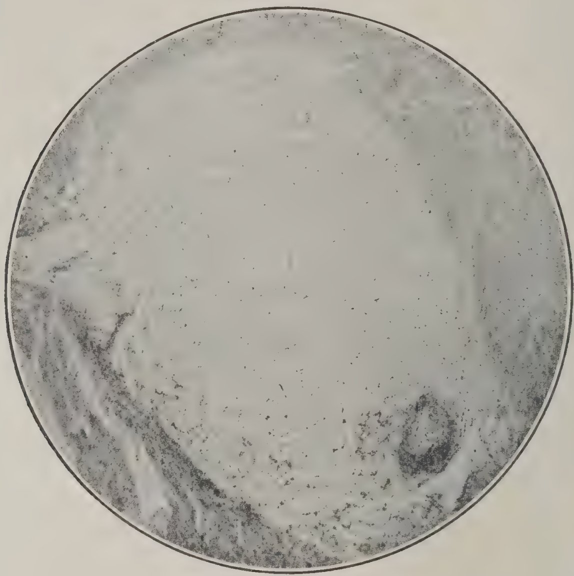

Fig. 67.—Fatty Infiltration of the Heart Muscle. A mass of fat can be seen embedded in the wall. (*Leitz obj. No. 3.*) (*From the author's private collection.*)

fat, and in part by the secondary atrophy of the specific cells due to pressure and imperfect nutrition.

Hydropic Degeneration.—This form of degeneration is characterized by the partial liquefaction of the cellular substance, resulting in the formation of clear vacuoles within the cell. The degeneration may take place both in the cytoplasm and in the nucleus. The nucleus may be so ballooned out as to resemble a little sac filled with colorless fluid. Hydropic degeneration is the first stage of colliquative necrosis, and is also found in vesication of the skin, inflammation, and in the cells of tumors. The cell and its nucleus stain badly, thus indicating that it is a retrograde process.

Colloid Degeneration.—Colloid is a semi-solid, translucent, homogeneous, and structureless substance, of a yellowish or brownish color. In general appearance it resembles stiff glue. Colloid is found normally within the follicles and lymphatics of the thyroid gland and in the pituitary body. It bears a general relationship, so far as external appearance goes, to mucin, hyalin, and amyloid. Chemically, it differs from these in some particulars. Its exact composition is unknown, but it is believed to be an albuminous body containing thyroidin.

The material does not swell up in water, is not precipitated by alcohol or acetic acid, and is stained orange-red by Van Gieson's method.

Pathologically (see Fig. 68), colloid is found in increased amount in certain cases of enlarged thyroid (colloid goitre), and in some tumors of the thyroid. Material resembling thyroidal colloid is at times found within the kidney (colloid casts), in cysts of the kidney or ovary, in the prostate and parotid, and in some carcinomata. Whether it is identical with true colloid is perhaps doubtful. Colloid and colloidal material must in all probability be regarded as products of epithelial cell activity.

Mucinous Degeneration.—Mucus is a homogeneous, transparent, slightly ropy material, the chemical constitution of which is not exactly known. Probably a number of substances, in general resembling one another, but differing slightly in composition, are included under the term *mucus*. The principal ones known at present are mucin and pseudo-mucin. Mucin contains nitrogen and sulphur, swells in water, is dissolved in alkaline fluids, is precipitated by alcohol and acetic acid. It is non-diffusible. Pseudo-mucin dissolves in water and is not precipitated by acetic acid. From both mucin and pseudo-mucin a carbohydrate

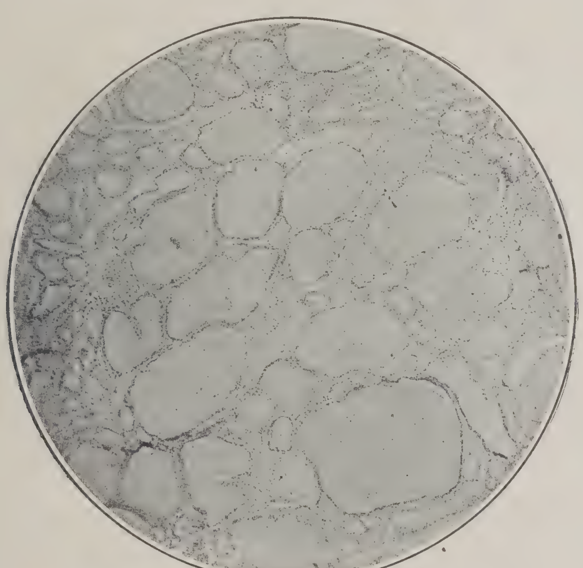

Fig. 68.—Excessive Production of Colloid in the Thyroid Gland—Colloid Struma. (*Leitz obj. No.* 3.) (*From the author's private collection.*)

may be obtained, indicating that they are to be regarded as glyco-proteid in nature.

Mucus is found normally as a secretion of mucous membranes and mucous glands, in joints, tendon sheaths, and bursæ, and forms the Wharton's jelly of the umbilical cord. In the case of the mucous membranes, mucus seems to be the special secretion of certain cells, called from their appearance "goblet cells."

These cells, when in an active condition, contain a clear, colorless, transparent, oval globule, which eventually is extruded upon the surface of the membrane or into the lumina of the glands.

Pathologically, mucus is formed in considerable quantities in various conditions. Thus, in inflammation of mucous membranes, there is an excessive production of mucus from the superficial epithelium and the glands. The number of goblet cells appears also to be increased. Not only so, but, unlike what occurs in normal secretion, the cytoplasm and even the nucleus of these cells undergo mucinous degeneration leading to complete destruction of the affected cells. The globules of mucin coalesce and we get a continuous sheet of stringy mucus covering over the inflamed surface (catarrhal inflammation). Pus cells, if present in the exudate, may in their turn undergo the same transformation. In such cases the presence of mucus in excess is to be regarded as protective in its nature, for, although non-bactericidal, mucus, by its mere presence, interferes with the action of pathogenic bacteria, and in the course of its excretion tends to flush out the diseased area. Mucoid degeneration is also not infrequently met with in the epithelial cells of tumors, especially carcinomata, involving the mucous membranes. In such cases large masses of carcinomatous tissue may be converted into mucin. Such tumors have, on section, a sticky, gelatinous appearance. The epithelial cells in large part disappear, and their place is taken by a fibrillar, loosely arranged material, which strikes a bluish tinge with hæmatoxylin. Pseudo-mucin is found in a large number of ovarian cystadenomata.

Mucinous degeneration is also to be observed in connection with mesoblastic structures. Here the intercellular substance loses its fibrillar character and is converted into mucin. This transformation occurs in connective tissue, cartilage, bone, bone-marrow, and fat. The connective-tissue framework of some sarcomata and carcinomata may show the change. Microscopically, in such cases we find bipolar or stellate cells, with long processes, floating in a loose, structureless, colorless matrix. The majority of nasal polyps are mucinous in character. In myxœdema, a curious disease due to inadequacy of the thyroid secretion, there appears to be a mucoid metamorphosis of the subcutaneous tissues.

Cholesterin.—Plates of cholesterin, apparently the product of degeneration, are found in atheromata, cysts, the walls of sclerotic vessels, old extravasations, and purulent exudates. In such cases it appears to be a by-product in the process of fatty degeneration. Cholesterin is found in the form of thin, rhombic plates, frequently having a small rhomb taken out of one corner.

Epithelial Hyalin.—A number of substances, bearing a general resemblance to colloid or hyaline material, have been grouped under this head. Probably they are not all of identical chemical composition. Warthin would restrict the term "epithelial hyalin" to the degeneration products of epithelial cells, which

resemble the hyalin of connective tissue in that they stain with fuchsin. In this category would come, therefore, the hyaline granules and globules found in carcinoma cells. These have been thought to be parasites, but are really due to degeneration of the epithelial cells.

Cornification.—Cornification occurs normally in the skin. Excessive cornification (hyperkeratosis) takes place exceptionally. The condition may be congenital, as in ichthyosis congenita, or acquired. Irritation of the skin of all kinds, mechanical or inflammatory, may result in hyperkeratosis. Familiar instances are warts, callosities, and corns. Cornification may also occur pathologically in parts of the body where it normally should not take place at all or only to a trifling extent. Thus, the ducts of the cutaneous glands may be affected and even blocked. Mucous membranes also, such as those of the mouth, vagina, urinary passages, middle ear, and mastoid cells, may on occasion be transformed into skin-like structures. Keratohyalin is also produced in certain tumors of the skin, brain, and meninges. In such cases the process is a true cell degeneration, the horny material being formed by the cells at the expense of their nuclei. The nuclei shrink and ultimately disappear.

Pigmentation from Cellular Activity.—The pigments found in the body are derived from various sources. They may be the result of cellular activity—*autochthonous* or *metabolic pigments;* they may arise from changes in the blood, with liberation and modification of the hæmoglobin—*hæmatogenous pigments;* they may be derived from the bile which has been absorbed into the tissues—*jaundice* or *icterus;* or they may be foreign material imported from without—*extraneous pigments.*

At the present moment we are concerned exclusively with the metabolic pigments. Pigment normally is found in many parts of the body in the form of yellowish, brown, or black granules within the cells or in the intercellular substance. Thus it is present in the deeper layers of the rete Malpighii of the skin, in the choroid and retina, in the hair, and in the ganglion cells of the central nervous system. It is found also in the connective tissue of the pia mater, the heart and other muscles, the kidneys and suprarenals. According to their chemical peculiarities, pigments are called melanin, hæmofuscin, and lipochrome.

The normal amount of pigment may be increased under certain physiological and pathological conditions. The pigmentation of the skin becomes more intense in certain regions during pregnancy. Sunburn results in increased coloration of the affected part. Freckles are a form of increased pigmentation. In Addison's disease there is an excessive formation of pigment in the skin and mucous membranes. The most extreme examples of pathological pigmentation are found in certain moles of the skin and in the melanotic sarcomata and carcinomata. In these growths the pigment lies both within the cells and in the intercellular substance in the form of fine brownish or blackish granules. Some of these tumors are coal black. In such cases the urine may contain substances

which turn black on exposure to the air (melanuria). In the examples cited it is believed that the pigment results from cellular activity of a special nature. Koelliker holds that in the skin the pigment is carried by wandering connective-tissue cells (chromatophores), which send processes between and into the epithelial cells, and there deposit their pigment. The source of these chromatophores is quite unknown. In what way the pigment is produced is also unknown. It would seem probable, however, that it is elaborated by the cells from albuminous substances.

Besides the melanin found in the cases just referred to, we have hæmofuscin, supposed to be identical chemically with hæmatoidin. It is found occasionally in the heart and in the unstriped muscle of the intestine.

Lipochrome is a pigment, or rather a class of pigments, of obscure nature, found in the corpus luteum of the ovary as a yellow-colored fat (lutein), and in the rare tumor known as the *chloroma*, which is of a pale greenish color. The so-called xanthoma of the skin contains a coloring matter belonging to the lipochromes. Ochronosis is a brownish or brownish-black pigmentation of the cartilages found in rare cases. The nature of the condition is unknown.

Glycogenous Degeneration.—Glycogen is a carbohydrate, an intermediate product in the conversion of starches into sugar. It is found normally in the liver, in the mucous membrane of the uterus, in the voluntary muscles and the heart muscle, in leucocytes, blood serum, and cartilage, and in most of the organs during embryonic existence. Glycogen is found in the tissues either in solution or as flakes or granules of hyaline appearance, lying within the cells or in the intercellular substance. It is soluble in water. When treated with iodine it stains a brownish-red. Unlike amyloid, it does not give the reaction with iodine and sulphuric acid. Amyloid, again, is not soluble in water. In examining tissues for the presence of glycogen, it is important to fix and examine the material immediately after death, as the glycogen is quickly transformed into sugar.

Pathologically, glycogen is formed in increased amounts in pus cells, in the leucocytes in certain cachectic conditions, and in the cells of some tumors, notably tumors of the kidney and suprarenal (hypernephromata), of the cervical portion of the uterus, of the testes, bones, cartilages, and muscles. In all these cases it is probably a result of cell activity.

INFILTRATIONS.

Amyloid, waxy, or lardaceous infiltration is the condition in which there is a deposit of a glassy, wax-like, homogeneous substance in the walls of the smaller blood-vessels. Almost any part of the body may be affected. Amyloid infiltration is most commonly met with in the spleen, kidneys, and liver; less often in the stomach, intestines, heart, lymph nodes, suprarenals, and pancreas; rarely in the muscles, uterus, ovaries, and respiratory tract.

The exact nature of the infiltration is not entirely understood. It occurs as a secondary disturbance in a variety of ailments, chiefly chronic and infectious, which are accompanied by grave disturbances of nutrition. Thus we get it most commonly in chronic tuberculosis, especially of the lungs, bones, and joints; in chronic suppuration, as in osteomyelitis, pyæmia, actinomycosis, glanders; in inveterate syphilis, in chronic dysentery, in prolonged lactation. Local amyloid infiltration of the kidneys is occasionally met with in chronic Bright's disease. The condition is also sometimes found in connection with leukæmia, carcinoma, and severe malaria. Ziegler is the authority for the statement that amyloid change may arise in the absence of previous disease. It can be produced experimentally. Czerny caused it in dogs by inducing long-continued suppuration with injections of turpentine. Krawkow produced it in rabbits and chickens by repeated injections, in increasing quantities, of broth cultures of the Staphylococcus pyogenes aureus and the toxin of Bacillus pyocyaneus. In these cases the condition appeared in from one and a half to two months. Thus it is evident that amyloid change is directly related to the disorder of nutrition resulting from chronic cachexia. The more intimate explanation of the process is still to seek. It has been suggested that the parenchymatous cells of the various organs are directly changed into amyloid material, but this is not supported by histological evidence. Amyloid is not found within the cells, but in the interstices of connective tissue and in the walls of blood-vessels. In the early stages it has been found just beneath the endothelial lining of the vessels. This, together with the fact that the vessels are so frequently picked out for the transformation, strongly supports the view, which is now quite generally accepted, that the amyloid, or, more probably, some precursor of it, is circulating in the blood and is precipitated in the walls of the vessels or in the perivascular lymphatics. Possibly we should take into account also the selective properties of the endothelial cells, and the influence of the tissue juices, which may combine with the amyloid precursors to form amyloid. We may perhaps here allude to the views of Von Recklinghausen and Czerny. The former has advanced the theory that the cells of the organs excrete a homogeneous substance, which coagulates in the tissue spaces into the characteristic amyloid deposit. Czerny found cells giving the microchemical reaction of amyloid in the pus and blood of animals which later presented amyloid change in the spleen. He therefore thinks that in the early stages the amyloid material is formed in local foci of suppuration and is carried to the internal organs by the leucocytes.

Chemically speaking, as Oddi and Krawkow have shown, amyloid is a compound of albumin and chondroidin-sulphuric acid. The latter substance is found normally in cartilage and all structures containing abundance of elastic tissue, especially in the blood-vessels. Amyloid does not contain phosphorus. It is practically insoluble in water, is unaffected to any axtent by acids or alkalies, and resists the action of the gastric juice and even of decomposition. There are

certain chemical tests which give characteristic reactions with amyloid material. To determine the presence of amyloid in organs removed at autopsy, thin slices are taken, the blood is removed by washing, and the material is allowed to remain in a solution of iodine in potassium iodide (iodine 1 gm., potassium iodide 2 gm., water 300 c.c.) for a few minutes. If for any reason the organs are alkaline, it is necessary to soak the tissue first in acetic acid. If amyloid be present, a mahogany-brown, translucent coloration is produced. Thin sections, stained by this method, when viewed under the microscope, show the amyloid as a glassy, transparent substance of a golden-yellow color. More striking still is the reaction produced with certain aniline dyes. Microscopic sections are placed for two or three minutes in a somewhat dilute watery solution of methyl-violet or gentian-violet. The sections are then washed in a weak acid solution, such as acetic or hydrochloric (two per cent), until most of the blue color is removed. If amyloid be present, portions of the tissue will assume a rose-pink color, which can also be made out very well under the microscope. The unaffected parts strike a dirty, grayish-blue tint.

Organs the seat of advanced amyloid change are usually enlarged, their edges somewhat rounded, and are much increased in consistency, so that they feel like India rubber. On section the amyloid material can often be made out as dots or streaks of a grayish, translucent appearance, or the surface of the organ may look as if smeared over with gelatin. Thin sections on being held up to the light appear to be pale gray and translucent.

In the spleen amyloid infiltration begins in the walls of the arteries, capillaries, and smaller veins, especially those within the Malpighian bodies. These become greatly enlarged and appear in the fresh state like grains of half-boiled sago; hence the term "sago spleen." Or, again, the condition may spread, involving the vessels and trabeculæ of the pulp, giving rise to a diffuse amyloid infiltration—"waxy," bacony, or lardaceous spleen.

In the kidneys the process begins in the walls of the interlobular arteries, afferent arterioles, and glomerular capillaries. The middle coats of the arteries are first and chiefly attacked. The glomeruli in time become largely converted into structureless, translucent nodules.

In the liver it appears first in the walls of the intralobular capillaries, mainly in the intermediate zone of the lobules, forming thick, homogeneous bands, between which are liver cells in all stages of atrophy and fatty degeneration.

Amyloid infiltration is always of serious import. It is met with only in most dangerous disorders and in the most advanced stages of them. It indicates, then, a bad prognosis where it can be made out. It also aggravates the primary disease by inducing atrophy and fatty changes in the cells with which it comes in contact, and causes marked circulatory disturbances, with all their consequences, through alteration in the lumina of the affected vessels.

Closely resembling amyloid in general appearance, although differing from it

in several important particulars, is *hyaline infiltration.* Like amyloid, this takes place in the walls of blood-vessels and in the interstices of connective tissue. It is also met with in inflammatory exudates. It is distinguished from amyloid by the absence of the iodine reaction and the rose-pink color when treated with aniline dyes. By Van Gieson's method it stains a deep red, while amyloid stains pinkish-yellow or brown. Again, hyaline infiltration is not so regularly distributed in the body as amyloid, nor does it have the same etiological relationship with suppuration and chronic cachexias. The close relationship which exists between hyalin and amyloid is shown by the fact that hyaline deposits are not infrequently found in organs the seat of amyloid change, and, conversely, amyloid, when introduced into the peritoneal cavity of experimental animals, loses its characteristic staining properties and becomes like hyalin.

Hyaline infiltration is found in the walls of sclerotic vessels, in the heart valves when the seat of chronic inflammation, in the connective tissue of the thyroid, ovaries and lymph nodes, and in the stroma of many tumors. Hyaline transformation of the glomerular tufts is often met with in the kidneys in chronic Bright's disease. The cellulo-fibrinous exudate in certain cases of pleurisy, pericarditis, and peritonitis occasionally undergoes this transformation. This may lead to great thickening of the serous membranes, so that they come to resemble cartilage ("icing" organs, "Zuckergussorgane"). In certain forms of coagulation of the blood, as, for example, in the formation of blood-platelet thrombi, the platelets become fused into a mass resembling hyalin. The so-called "hyaline bodies," or Russell's "fuchsin bodies," are homogeneous, globular masses of varying size, either single or aggregated in clusters. They are found both within and without the cells in glandular proliferations of the gastric mucosa and in malignant tumors. They strike a red color with acid fuchsin and dark blue with the Gram-Weigert stain. By some they have been regarded as of parasitic nature. This, however, is now believed to be erroneous.

Certain changes in glia cells sometimes produce masses which stain black with the Pal-Weigert method, red with Van Gieson's stain, and bright blue with Weigert's fibrin stain, after fixation in Zenker's fluid. Barker has shown that the material in question forms within the glia cells.

So-called amyloid bodies or concretions are also met with in the prostate (Fig. 69), hypophysis, central nervous system, and lungs. Some are homogeneous and others laminated. In the latter case it is not uncommon to find in the centre of the concretions cell débris, indicating that in some cases at least the process is probably a degenerative one, resulting from the cutting off of desquamated cells from their nutritive supply. Occasionally, but by no means invariably, such bodies give the reaction for amyloid. Their exact relationship to amyloid, hyalin, or colloid cannot as yet be stated.

The exact nature of the hyaline change is still unknown. When occurring in the connective tissue of certain organs, especially that of the conjunctiva, and in

inflammatory exudates, hyaline material appears to be more of a degeneration than an infiltration. Theoretically, the connective-tissue elements may be transformed into hyaline material containing no nuclei, or the hyalin may be a secretion from the connective-tissue cells. In some cases the process appears to be both a degeneration and a deposit, the interstices of the connective tissue being first filled with a clear, homogeneous material, into which the cells gradually fuse.

Calcification and Analogous Conditions.—Under certain conditions there may be a deposit in the body of crystalline, amorphous, or granular salts, derivatives of lime or uric acid. This is called *petrifying infiltration.* The deposition of lime salts is usually termed calcareous infiltration or *calcification.* This is of not infrequent occurrence. The precipitation of these substances may take place into tissues or structures which are normally part of the body, into structures which

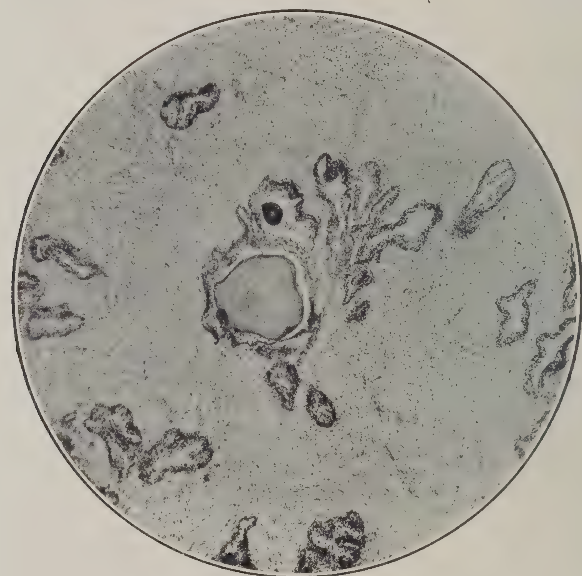

Fig. 69.—Corpora Amylacea in the Prostate. (*Leitz obj. No. 3.*) (*From the author's private collection.*)

are separated from their normal relationships, or into foreign bodies imported from without. In the last two instances we speak of the formation of *concretions* or *calculi.*

Calcification occurs as a normal change in the formation of bone from cartilage. In advanced life lime salts are deposited with great regularity in the costal cartilages, in the cartilages of the larynx, and in the walls of the arteries. It is generally believed that this is due to certain involutionary changes in the bone which occur in old age, the lime salts being reabsorbed and deposited elsewhere in the body. In some cases of osteoporosis and osteomalacia the salts are depos-

ited in apparently normal tissue, as in the lungs, kidneys, and gastric mucosa. This process is sometimes referred to as *metastatic calcification* or *lime metastasis.*

Calcification almost never occurs in normal tissues. It is practically always preceded by cloudy swelling, fatty degeneration, hyaline transformation, caseation, or necrosis. Calcareous deposit occurs not infrequently in connective tissue which has become sclerosed or hyaline; for example, in arteriosclerosis, in the heart valves when the seat of chronic endocarditis, in chronic pleurisy, empyema, and chronic pericarditis, and in the thyroid gland. It occurs in tumors, such as uterine fibroids. It is found in necrobiotic and necrotic areas, as in caseation or coagulation necrosis, in old inflammatory exudates, in thrombi, and in the capsules of animal parasites. Calcareous infiltration may also take place in dead ganglion cells of the brain in cases of shock and softening. It is met with in the renal epithelium in the necrosis resulting from anæmia and from intoxications, such as those due to corrosive sublimate, aloin, and bismuth. Foreign bodies, such as a dead fœtus (*lithopædion*), catheters, bullets, pessaries, etc., may become incrusted with lime salts.

The exact chemical reactions which occur in the formation of lime deposits are still more or less a matter of debate. The lime is present in the tissues in the form of carbonate and phosphate. When strong mineral acids are added to the material, there is an evolution of carbonic dioxide. The problem is chiefly indicated in the following questions: How do the lime salts enter the system? In what form do they exist? What brings about their precipitation?

The physiological infiltration of certain tissues with lime salts which occurs in old age is with great probability to be referred, at least in the main, to a transference of the lime from the bones to other parts. The fact, however, that we find calcification so often in early life would indicate that there must be another explanation for its occurrence. We have to believe, then, that the process is intimately bound up with the body metabolism and that the ultimate source of the lime is in the food. A farther point is that calcification does not occur except in tissues that are in a more or less advanced stage of degeneration. There appears to be in such tissues some chemical substance which determines the place of the deposit. In rabbits and ruminants, whose food abounds in lime salts, it is a very common thing to find lime deposits at points where there is local death of tissue. Inasmuch, however, as such local death occurs frequently in human beings without calcification supervening, we have to conclude that an excess of lime salts in the food is necessary. These salts, no doubt, are carried throughout the body in the blood and lymph, presumably in a soluble form. When they reach the degenerated tissues it is held by some that they are acted upon by the phosphoric acid and nascent carbonic dioxide and thrown down in an insoluble form, or, what is perhaps more likely, they first combine with the fats in the part to form soaps, which are in their turn decomposed. The salts are thus laid down in solid masses both within the specific cells of the

tissue and in the intercellular substance, and may abound to such a degree as to render the part hard like stone. Microscopically, the lime appears as fine, refractile granules, which are dark and somewhat opaque by transmitted light and white by reflected light. In sections stained by hæmatoxylin the granules strike a purplish-black color. The condition is essentially one of deposition of mineral matter, and hence is often termed *petrifaction*, as contradistinguished from *ossification*, in which, together with the deposition of salts, there is a formation of new tissue.

The deposition of salts of uric acid is of great practical importance. In the disease known as gout they are laid down in the articular cartilages, ligaments, tendons, and tendon sheaths, the subcutaneous connective tissue, and in the kidneys. The material is deposited in the form of needle-shaped crystals, sometimes truncated, in the intercellular substance of the cartilage. At first the cartilage cells do not suffer, but later the deposit may be so great that the cartilage becomes opaque, looking like chalk by reflected light. In the most extreme cases the affected part may actually necrose. As might be expected, the presence of so much foreign material, and that of an irritating kind, leads to inflammation of the structures involved. In gout there is evidently a marked disturbance of metabolism, in which unusually large quantities of uric acid are formed in the blood. Uric acid is a very insoluble substance, and it has been suggested by Roberts that it exists in the blood as a quadriurate soluble in water. This is broken up in part into sodium biurate, which is the substance precipitated.

The uric-acid infarcts found in new-born infants deserve a word. They are observed usually in the first two weeks of life, rarely in the fœtus, and appear as yellowish- or reddish-white striated marks in the papillæ. They are due to the deposition of urates in the uriniferous tubules, and probably indicate a slight disturbance of metabolism.

Calculi or *concretions* are rounded, nodular, or branched masses of mineral matter formed in the tissue spaces, in the lumina of vessels, in ducts of glands, and in cavities lined by mucous membrane, by precipitation from the fluids or excretions of the body. Some of the so-called "free bodies" are composed entirely of organic material, such as certain of the "amyloid" concretions in the prostate and central nervous system, but as a rule in the organic matrix there is a deposit of insoluble salts.

Brain sand is composed of small calcareous masses of this kind, and is found normally in the pineal gland, in certain tumors of the dura and pia mater, and of the choroid plexus, hence called psammomata. Similar concretions are at times found in the various cavities of the body. Such are the petrifications of old thrombi (arterioliths, phleboliths), calculi in the hepatic and urinary passages, concretions in the pancreatic and salivary ducts, in the nasal (rhinoliths) and respiratory passages (broncholiths), in the external auditory meatus (otoliths), fecal accumulations in the intestines, preputial stones.

Gall stones are among the commonest forms of calculi. They are met with usually after middle life, and are much more frequent in women than in men (2 to 4 : 1). According to their chemical constitution we may recognize four kinds of biliary calculi—cholesterin, bilirubin-calcium, pigmentary, and calcium carbonate. Various combinations of these may occur. Pure cholesterin calculi are rare. They are often solitary and may form a complete cast of the gall bladder. They are light-colored, often somewhat greenish, are hard, and break with a crystalline fracture. They are made up of radial and concentric laminæ. Mixed calculi of bilirubin or biliverdin and calcium salts are more common. They vary greatly in size, are often very numerous and faceted, are hard, and of a dark brownish color. Pigment calculi are small, irregular, and friable.

The *modus operandi* in the formation of gall stones has been fairly well worked out by Naunyn and others. The first requisite for the formation of biliary calculi is an albuminous matrix, and then an abnormal biliary secretion. Catarrh of the biliary passages provides the first condition. This may be brought about by stasis of the bile, such as may be induced by sedentary habits, obesity, tight lacing, and too long intervals between meals. This leads to slight irritation of the mucosa with the liberation of an albuminous secretion and the desquamation of some of the lining cells. Or the same result may be brought about by intestinal or general systemic disturbances, resulting in invasion of the biliary passages by bacteria. The Bacillus coli, the Bacillus typhosus, the staphylococcus, and the streptococcus have been found in the bile passages, and are known to persist there on occasion for months. Both the colon and the typhoid bacillus have been found in gall stones, and typhoid fever is now looked upon as an important etiological factor. Biliary calculi have also been produced experimentally by injecting bacteria into the gall bladders of animals. The desquamated cells and albumin tend to fuse together and form the nucleus in and about which the various salts will be deposited. The source of the cholesterin and bilirubin-calcium is not entirely clear, and conflicting opinions have been expressed. Probably Naunyn's view is most widely held. According to this, cholesterin is formed *in situ*, being produced by the diseased mucous membrane. Increased acidity of the bile appears to have something to do with it. Bilirubin-calcium is not a normal constituent of the bile. Bile salts seemingly have a retarding influence on the formation of bilirubin-calcium, but this inhibitory power is counteracted by the presence of albumin, so that the precipitation of this substance is similarly traceable to the condition of inflammation. The deposition appears to take place, not according to the usual rules of crystallization, but under the influence of the albumin the mineral matter is precipitated in the form of plates. Owing apparently to variations in the local condition of things, the calculus is built up gradually layer after layer, and in radial form. The great variation in the composition of gall stones would, however, seem to indicate that there are other factors besides those mentioned. Probably local

disturbance of the hepatic functions and disorders of systemic metabolism are of importance.

Urinary Calculi.—These calculi may be found within the tubules of the kidney, in the kidney pelvis, and in ureter, bladder, and urethra. Not infrequently they are formed in one place and carried by the flushing-out action of the urine and muscular activity to some more remote part. Calculi are produced in the kidney (renal calculi) or in the bladder (vesical calculi). By their presence they frequently cause obstruction to the outflow of the urine and inflammatory disturbances, with even destruction, of the urinary organs.

The chemical constitution of urinary calculi depends, on the one hand, on the composition of the urine, which in its turn is dependent on the general metabolic processes of the body; and, on the other, on certain chemical changes occurring in the urine after its secretion by the kidneys. As in the case of biliary calculi, we have to recognize both local and general disorders of metabolism. Chemically speaking, urinary calculi may be divided into the following forms: (1) The *uratic;* (2) the *phosphatic;* (3) the *calcium carbonate;* (4) the *calcium oxalate;* (5) *cystin;* (6) *xanthin.*

Calculi are built up gradually, and, owing to variations in the conditions, may present a different composition in different parts, or several calculi of different composition may be found in the same patient.

The important element in the production of urinary calculi is the disturbance of the general metabolism. This cannot act, however, unless the local conditions are favorable. As we have seen in the case of biliary concretions, an albuminous medium is a necessity. The work of Ebstein, Posner, Naunyn, and Studensky, among others, has shown conclusively that albuminous fluids have the power of determining the precipitating of the crystalline salts from their solutions, much in the same way as in the formation of the hen's egg, where the albumin has the power of separating out carbonate of lime from solutions of calcium salts. To bring about the excretion of albuminous material, the chief factor is of course inflammation. This may be produced by stagnation of urine, alterations in the composition of the urine, local bacterial invasion, fermentative processes, and the like. The lining cells desquamate to some extent and act as a sort of nucleus. As in the case of biliary calculi, catarrhal inflammation is of great importance; but as every catarrh does not result in calculus formation, we must believe that other factors are at work. These are not altogether clear, but are probably to be looked for in some abnormality of general metabolism. Hyperacidity of the urine, due to an excess of uric acid, is a predisposing cause of the precipitation of uratic salts and oxalates. When urine becomes alkaline, as from retention and the activity of certain bacteria, ammonio-magnesium phosphate is formed and may be thrown down. Incrustations of phosphates occasionally are formed upon foreign substances which get into the bladder, such as bits of catheters, slate pencils, hairpins, tooth brushes, etc.

Cystin calculi originate in disturbances which take place outside of the urinary organs. Such calculi, which are rare, are due to abnormal decomposition of albuminous substances in the intestines brought about by bacteria. Cystin calculi are yellowish, soft, and waxy.

Xanthin calculi are also rare. They are found in the bladder, and are reddish in color, soft, and friable.

In cattle, calculi formed of silicates may be met with.

Fecal calculi are composed of inspissated fecal matter infiltrated and incrusted with lime salts. Constipation and the presence of intestinal diverticula favor their formation.

Salivary and pancreatic calculi are also composed of lime salts, usually the carbonate.

Glycogenous Infiltration.—Infiltration with glycogen is found especially in the case of diabetes mellitus. In this disease both glycogen and sugar are produced in greatly increased amounts. The glycogen may be demonstrated in the leucocytes, blood plasma, liver, and kidneys. In the last-mentioned organs the deposit occurs chiefly in the epithelial cells lining the loops of Henle. It is usually found in hyaline-looking droplets near the nuclei, and may be recognized by the before-mentioned tests. Experimental diabetes, produced by the removal of the pancreas, is followed similarly by a deposit of glycogen in the leucocytes, liver, and kidneys.

Pigmentary Infiltration.—We have above referred to a form of pigmentation which is due to metabolic changes in the cells themselves. This has properly been regarded as a true degeneration. There are other forms of pigmentation, however, which, while in some instances the result of disturbed metabolism, are, so far as the affected cells and tissues are concerned, the result of causes operating outside of them. Here the pigment which is produced in one part of the body is carried by the blood or lymph to other regions, where it is deposited as so much foreign material. This is pigmentary infiltration. In this class are to be considered the pigmentation which results from breaking down of the red blood corpuscles—*hæmatogenous pigmentation,* and that due to the accumulation of bile in the tissues—*biliary pigmentation.*

In the first-mentioned variety the pigmentation is due to the deposition in the tissues of coloring matter derived from hæmoglobin. This may occur as the result of hemorrhages or thrombosis, where the blood cells become, as it were, extravascular, and undergo retrograde changes, or it is due to the solution of the hæmoglobin in the plasma and the formation of granules of pigment in the blood.

Hæmatogenous pigments occur in two main forms—*hæmatoidin* and *hæmosiderin.* Hæmatoidin is found in the form of yellowish or brownish granules, or as reddish rhombic or acicular crystals. It responds to Gmelin's test and is believed to be practically if not quite identical with bilirubin. It does not contain

iron, is insoluble in water, ether, and alcohol, but soluble in alkalies and chloroform. It is found more particularly in connection with large hemorrhages, especially when they occur into some cavity. In such cases the blood corpuscles are to a comparatively slight extent acted upon by living cells and the supply of oxygen is relatively scanty.

Hæmosiderin occurs in yellowish-brown or brown granules, usually within the cells, but also free in the tissue spaces. It is insoluble in water, and differs from hæmatoidin in that it contains iron. This may be demonstrated by Perl's test. If a microscopic section containing this pigment be treated with a three-per-cent solution of potassium ferrocyanide and then with a weak solution of hydrochloric acid, the iron-containing granules take on a bright blue color, owing to the formation of Prussian blue. If treated with hydrogen sulphide, the granules turn black. Hæmosiderin is formed where the blood cells are exposed in small quantities to the action of living cells and oxygen. Therefore we find it at the site of small effusions of blood, at the margins of larger ones, in small thrombi, and in organs the seat of chronic passive congestion.

Hæmatogenous pigmentation arises, in general, wherever there is extravasation of blood. The pigmentation is to be attributed to physical and chemical changes in the red corpuscles when out of their normal environment, resulting in a transformation of the hæmoglobin. The extravasation of the blood may be due to injury, thrombosis, rupture of vessels, or degenerative changes in the vessel walls, the result of the deleterious action of mineral, bacterial, or other toxins. After an extravasation of blood the red cells are to some extent broken down, and there is an attempt on the part of the cells of the body to remove the débris. The process appears to be as follows: Some of the unaltered red corpuscles get back to the circulation by means of the lymphatics; some fragment and disintegrate into brownish or reddish particles, containing hæmoglobin; some, again, lose their hæmoglobin, which dissolves out in the plasma, and the albuminous framework of the cells ultimately breaks down. Part of the liberated hæmoglobin passes in the circulation to the organs of excretion, and is eliminated in the urine as methæmoglobin and urobilin. The remainder, together with the remains of the red corpuscles and other detritus, is picked up by the phagocytes or carried by the lymph to various organs, such as the regional lymph nodes, spleen, liver, and bone-marrow, where it is acted upon by the cells of the part or by the oxygen, and deposited in the form of yellowish or brownish granules. The color changes from black to brown, greenish-yellow, and yellow, as may be observed in the familiar instance of the common "black eye." Here the changes in color furnish an external indication of the chemical transformation which occurs in all such cases. Ultimately, the hæmoglobin is transformed into hæmatoidin, hæmosiderin, or both.

Similar pigmentation occurs from the destruction of the red corpuscles in the circulating blood. This is met with in such conditions as septic infection, per-

nicious anæmia, leukæmia, and malaria, in poisoning with certain substances, like potassium chlorate, antipyrin, toluylenediamin, fungi, and some bacterial toxins; and after the introduction, into the circulation of one animal, of the blood of another of a different species. In such cases the hæmoglobin may be liberated into the plasma (*hæmoglobinæmia*) and excreted by the urine, which thus becomes brownish-red or dark red in color (*hæmoglobinuria, methæmoglobinuria*).

Up to a certain point the organs of the body directly concerned in the transformation are able to deal with the increased amount of hæmoglobin and its derivatives which reaches them, but in some cases so much pigment is liberated that the blood-destroying organs become highly colored, or even the whole body may become affected. This is the case in a curious and rare affection, called by Von Recklinghausen *hæmochromatosis*, in which hæmosiderin is deposited in all the organs and tissues of the body. We have met with one case of this, in which the skin and mucous membranes were of a dark leaden hue. The condition is associated with fibroid changes in the liver (cirrhosis), pancreas (diabète bronzé), or in both. The liver is able to change part of the pigment which reaches it into bilirubin, and excretes it in the bile, but any excess in the amount which it is able to transform is deposited in the various tissues, and gradually eliminated by the kidneys as urobilin.

In the liver hæmatoidin is usually deposited in the parenchymatous cells toward the centre of the lobules, while hæmosiderin is laid down more at the periphery. In extensive hæmosiderosis the iron-containing pigment is laid down in the interstices of the connective tissue of the portal sheaths as well. In the spleen, lymph nodes, and bone-marrow the pigment is chiefly found in the endothelial cells lining the blood-vessels. In the kidneys it is to be found in the secreting cells lining the convoluted tubules, in the endothelium of the vessels, and in the lumina of the tubules.

In another class of cases the pigment is produced in some other part of the body by disturbed metabolism, and is then carried by the lymph stream or by leucocytes to the pigmented part. An example of this is the transferrence of melanin from a necrosing melanotic sarcoma to the spleen, lymph nodes, and kidneys. The pigment may appear in the urine, and casts of melanin are sometimes to be found in the kidney tubules.

The second main form of pigmentary deposit is the biliary. In certain cases of obstruction to the free excretion of bile, it enters the blood and lymph and leads to a yellowish or sometimes greenish discoloration of the whole body. The pigments of bile are produced in the liver and are derived from the hæmoglobin of the blood, hæmatoidin and bilirubin being identical chemically. Under normal conditions the bilirubin formed in the liver is passed out in the bile into the intestine, where, after effecting certain changes in the food stuffs, it is in part evacuated with the fæces. Part of it, however, is absorbed through the intes-

tinal mucosa into the blood. Here it undergoes some transformation, the exact nature of which is quite unknown, but eventually it appears in the urine in the form of urobilin.

Any condition which interferes with the free discharge of the bile from the liver and bile passages will give rise to *jaundice* or *icterus*. Such causes may be at work in connection with the larger bile passages or within the liver itself. In cases of jaundice not only are all the structures of the body stained with bile, but the bile passes out in the urine, in severe cases causing it to assume a dark brownish color. Obstruction of the bile-ducts may be due to catarrhal inflammation of the mucous membrane, impacted calculi, the pressure of adhesions, scars or tumors, abscesses, enlarged lymph nodes, cirrhosis of the liver, and tumors within the liver. In some few cases, as in acute yellow atrophy of the liver, some toxin, apparently of an infectious nature, seems to be at work, without gross evidences of obstruction to the outflow of bile.

When the obstruction is complete no bile reaches the intestines. Consequently, the fæces become pale and clay-colored and very foul from abnormal fermentation. Intestinal digestion is, of course, interfered with. In bad cases delirium, convulsions, coma, and all the features of a profound toxæmia may supervene.

The obstruction to the discharge of the bile leads to dilatation of all the bile-ducts and of the finer bile capillaries within the liver itself. These latter may rupture and the bile may enter the blood directly. Ordinarily, however, it passes into the lymph channels and gets into the circulation by way of the thoracic duct. The liver cells become pigmented, owing to the impossibility of their getting rid of their secretion. All the organs and tissues of the body assume a yellowish or greenish tinge, and under the microscope solid masses of brownish or yellowish pigment in granular form can be recognized, especially in the lymph nodes, spleen, and bone-marrow. In the more persistent and severe forms of jaundice the various organs may contain bilirubin in solid form, or, rarely, in rhombic or acicular crystals. It should be remarked also that the presence of biliary acids in the blood leads to breaking down of the corpuscles and liberation of the hæmoglobin. This increases the work of the liver and provides more material to be converted into bilirubin. Thus a vicious circle is the result.

Besides the obstructive form of jaundice just described, there is another very important type, due to the destruction of the red blood corpuscles in the circulating blood. This may be the result of a variety of causes, the most notable being the infections and intoxications. Among these are septicæmia, yellow fever, inhalation of ether and chloroform, snake bite, transfusion of blood, and the exhibition of toluylenediamin. This form of jaundice was formerly termed *hæmatogenous,* under the impression that the bilirubin was produced in the blood. We know now, however, from the experiments of Naunyn and Minkowski, that the liver is essential to the production of bilirubin, so that the more correct nomenclature would be *hæmo-hepatogenous* jaundice. In such cases there must be an increased

formation of bilirubin in the liver, and this material then makes its way into the general circulation. How this occurs is not certainly known, but some think that the main factor is a catarrhal inflammation of the smaller bile ducts and capillaries in the liver.

Allied to jaundice is the sallow, earthy tint of the skin found in cases of constipation and cachexia. Here, there may be an absorption of urobilin from the intestine into the blood, and a moderate grade of disintegration of the red corpuscles due to toxæmia.

Deposition of Foreign Substances.—Foreign material may enter the body from the external world and be deposited in the tissues. Such substances may reach the interior of the body in three ways—by the skin, by the alimentary tract, and by the lungs.

Perhaps the commonest foreign substances introduced through the skin are Indian ink and certain aniline colors employed in the process of tattooing. Some of the introduced pigment in these cases remains in the minute scars that form, while some is carried away and deposited in the nearest lymph nodes, which in their turn become pigmented. Explosions may drive particles of gunpowder, coal dust, or dirt into the skin.

The chief substances that enter the system through the alimentary tract are lead, arsenic, copper, and silver. The prolonged exhibition of arsenic leads to a brownish discoloration of the skin. In chronic lead-poisoning the lead is deposited in the form of a sulphide along the margin of the gums. Copper leads to a greenish pigmentation of the gums. In former days salts of silver were extensively used in medical practice, especially for certain nervous affections. After the prolonged use of silver, the metal, presumably in the form of an albuminate, is deposited in the form of brownish or blackish granules in the tissues, which assume a dark leaden gray color (*argyria*). The silver is laid down principally in the skin, in the kidneys, in the intima of the vessels, in the serous membranes, and in the choroid plexus of the brain.

The inhalation of foreign material leads to a deposit of the inspired substance in the lungs (*pneumonokoniosis*). The most usual pigment thus laid down is coal dust (*anthracosis*); next to that, particles of stone (*chalicosis*, *silicosis*); and next, iron (*siderosis*). A variety of other substances may on occasion be deposited, as cotton, paper, flour, iron ore, tobacco, ultramarine blue.

When any of the substances mentioned are inhaled, a portion of the dust is entangled in the mucous membrane of the nasal passages, and to some extent it lodges in that of the upper respiratory passages, from which localities it is gradually eliminated by the secretion of the nose and by the act of coughing. If the amount inhaled be not excessive, this may suffice to get rid of all the foreign matter, but as a rule those who are subjected to such unwholesome conditions are operatives who must continue for prolonged periods breathing impure air. In such cases the ordinary means referred to are ineffective, and the for-

eign material reaches the lungs. It seems fairly established now that inhaled dust does not reach the alveoli of the lung directly, for physical reasons, and we have to seek some other explanation for the occurrence. If we take, for example, the case of the inhalation of coal dust, as it is met with in coal miners or those who live in smoky cities, we find that the excess of coal dust that cannot be eliminated is deposited on the mucosa of the upper respiratory passages, where it sets up a certain amount of irritation, resulting in cough and slight catarrhal inflammation. Phagocytes are attracted to the part, pick up the pigment, and carry it along the lymphatics to the recesses of the lungs, where it is deposited in the alveolar walls, the interlobular septa, and in the lower layer of the pleura. In all cases the process follows the course of the lymphatics. From the lungs the pigment is carried to the peribronchial nodes, which become coal-black in color and gritty. As a result of the irritation produced in the tissues by the foreign material, chronic inflammation is set up, with the formation of connective tissue. This results in hardening of the lung, especially along the course of the bronchi and the various septa. Coal dust is relatively innocuous, but other substances, such as iron, steel, or marble, are much more irritating and lead to extensive induration of the lung (*chronic fibroid pneumonia*).

In very extensive grades of the affection the lungs may become hard, heavy, and may grate under the knife. The lungs may even be unable to retain the great quantities of coal dust which reach them, so that the coal reaches the general circulation. This takes place either from the dust passing through the entire thickness of the vessels and mixing with the blood, or from the softening of anthracotic lymph nodes, with discharge of their contents into some large vein. The dust may also get into the general blood stream by passing along the lymphatics. Coal dust may thus in time be deposited in the liver, spleen, or bone-marrow. Welch has described a case in which so much coal was deposited in the liver as to give rise to a form of cirrhosis (*cirrhosis anthracotica*).

The lungs of new-born and young infants are devoid of this coal pigment, so that its absence, or the amount of it when present, gives us approximate information as to the length of time a person has lived, and therefore may be of some value in medico-legal cases.

The great importance of the inhalation of dust in connection with the health of workers in certain industrial occupations has led most civilized governments to enact laws looking to the providing of efficient ventilation, and in some cases to enforcing the use of proper respirators.

NECROBIOSIS AND NECROSIS.

Death of cells may be gradual or sudden in its onset. *Necrobiosis* (indirect necrosis) is a term coined by Virchow to designate that form of death which comes on slowly, the result of slowly acting causes. *Necrosis* (direct necrosis) is

immediate death. Both terms apply to a local condition, in contradistinction to death of the body as a whole—*somatic death.*

In the case of necrobiosis, the death of the part is preceded by some retrograde metamorphosis, such as atrophy, cloudy swelling, fatty degeneration, mucoid or hydropic degeneration, or by some pathological infiltration. In direct necrosis, or, as it is usually termed more shortly, necrosis, death is rapid and is not preceded by any pathological changes in cellular structure. It is not always easy to draw a hard-and-fast line between these antecedent degenerative changes and the resultant necrobiosis, or between necrobiosis and necrosis. Still it is well to keep the ideas distinct in our minds. The preceding degenerative processes occur so gradually and are so characteristic in their appearances that it is usually thought better to class them by themselves, and to regard them as the causes or, perhaps more correctly speaking, the precursors of necrobiosis, rather than the necrobiosis itself. Necrobiosis usually ends in necrosis, and for practical purposes may be regarded as an incomplete or slowly progressive necrosis.

In a sense the normal retrogression of cells incident to katabolic processes and the renewal of tissue is a physiological necrobiosis. It is not pathological, for it does not interfere with function, the dead cells being simultaneously replaced by new cells of like kind. It may, however, be, and often is, pathological when it takes place independently of the needs of the organism and leads to more or less functional disturbance in the part.

The causes of necrobiosis are practically the same as those of necrosis—lack of nutrition, infections and intoxications, traumatic, chemical, thermal, and mechanical influences. Any of these may act separately, or two or more may be combined. In general, we may say that deleterious agencies of slight grade, acting over prolonged periods, are more apt to produce necrobiosis than necrosis.

Necrobiosis may, no doubt, in slight grades, be perfectly recovered from, but many cases go on to complete necrosis. If, for instance, necrobiosis has followed albuminous degeneration, simple necrosis follows; if there has been antecedent fatty degeneration, soft caseation results; if hydropic degeneration preceded, colliquative or liquefaction necrosis is the consequence. The results of necrobiosis are in most cases those of necrosis, as one might expect.

Microscopically, the cells undergoing necrobiosis present, in addition to the retrograde manifestations which may have been there, karyorrhexis of the nuclei, with more or less karyolysis. This passes on into actual disintegration of the cells.

Necrosis, then, is death of a cell or a group of cells while they are still a part of the living body. In a pathological sense it includes all those conditions variously known as gangrene, mortification, sequestration, abscess-formation, ulceration, and caries. By surgeons, however, these terms are not generally used synonymously. In surgical parlance "gangrene" and "mortification" are usually taken to apply to death of the soft tissues, while death of bone is usually called

"necrosis." It should be mentioned also that gangrene, mortification, and sequestration always imply death of a part *en masse*. Gradual, almost imperceptible disintegration, or molecular death, is called *ulceration* in the case of the soft tissues, and *caries* in the case of bone.

As we do not know what constitutes cell life, the true nature of necrosis, or cell death, is in a large measure a sealed book to us. The exact change in the constitution of the cell which indicates the passage from life to death, and the time at which it occurs, are beyond our ken. The methods of hardening, preserving, and staining tissues in vogue at the present time suffice to give us fairly accurate information as to the state of the cells at the time the tissues were placed in the solutions. Of course the cells are killed by such methods, so that we are always studying dead material; but what we believe to be normal cells, under such circumstances, appear to be so different from others that we are able to infer, with some approximation to the truth, that certain cells were dead while still connected with the living body. In all cases, however, we are studying post-mortem or post-necrotic appearances, rather than the changes immediately dependent on the necrosis.

The exact chemical changes that underlie necrosis are unknown. Histologically, necrotic cells show minute changes both in the nucleus and in the cytoplasm. The nucleus apparently breaks up into fragments (karyorrhexis), a form of disintegration shown by Schmaus and Albrecht to be preceded by a peculiar transposition of the chromatin threads. This gives place to dissolution of the nucleus (karyolysis). The cytoplasm loses its finer structure, becomes more hyaline and opaque, and possibly vacuolated. Ultimately, such cells may fuse into an indistinguishable, structureless mass or may liquefy. In necrotic cells the nuclei stain badly as a rule, and seem to be fading away. In some instances, however, the nucleus contracts and stains more deeply than normal (pyknosis). Thereupon, the cell disintegrates or fragments, and particles of chromatin are liberated, to be disseminated throughout the necrotic area. As a result of this, the dead tissue, at least in the earlier stages, may stain more or less diffusely blue with hæmatoxylin. Finally, the whole of the cellular material is converted into a granular débris.

The causes of local death of tissue are very various. They, however, may in their essence be reduced to two—lack of nutrition and direct trauma.

Traumatic insults, by crushing or tearing the cells, lead directly to death of the part. Or, indirectly, injuries to the blood-vessels may interfere with the adequate supply of blood to a region, and thus necrosis results. Probably in most instances we have to take into account not only direct influence of the injury upon the cells, but also more or less disorganization of the ordinary means of circulation. Wherever the blood supply is absolutely cut off, necrosis is inevitable. Again, cells which are dislocated from their normal environment are very apt to undergo degeneration and necrosis. Injuries do not always produce

these effects at the exact spot where the injury has taken place. Thus, a cart-wheel passing over a limb may produce extensive laceration of the soft muscles, while the skin remains intact. Or a crushing injury to the trunk may result in tearing of the liver or spleen without any external manifestations. In such cases, if the patient live, necrotic changes in the damaged structures will supervene. Again, severe blows upon the head may lead to necrosis of the ganglion cells of the brain.

Injury to an artery may be from laceration, pressure, or traction. Should the intima be ruptured, a thrombus forms at the site of injury, with ultimate blocking of the vessel and necrosis of the supplied area. This is often serious, for the tissues are sometimes so deteriorated by the injury that a collateral circulation cannot be formed.

To be classed with mechanical trauma are heat, cold, and caustics.

Tissues subjected to a temperature of from 54° to 68° C. for a short time will undergo necrosis. The effects produced by heat depend on its intensity and the length of time during which it is operative. Take, for example, a limb. The least serious result is an active congestion of the part with slight inflammation (burn of the first degree). If the part be exposed somewhat longer, the superficial epidermis is elevated into blisters (burn of the second degree). In this case there is necrosis of the epidermis, which is detached in parts from the underlying tissues, owing to the accumulation of serous fluid. The cells may show hydropic degeneration. Or, the destruction of substance may extend below the skin (burn of the third degree); finally, the whole structure may be charred (burn of the fourth degree). Heat acts by coagulating the albumin of the cells which come under its influence.

Cold has an identical effect, the result depending on its degree and on the length of time the part is exposed to it. Cohnheim produced gangrene of a rabbit's ear by subjecting it for a short time to a temperature of 16° C. Freezing will produce extensive gangrene, especially in those cases where the circulation is not restored gradually and stasis results.

The *x*-rays produce extensive and very obstinate burns in some cases. This is not the effect of heat, but rather of some influence of the chemical rays, possibly, as has been suggested, upon the nerve endings.

Caustic substances, acids, alkalies, acid nitrate of mercury, chloride of zinc, will cause death of the structures to which they may be applied.

The most numerous cases of necrosis are to be traced to defective nutrition. In this connection interference with the circulation is the most important single factor. The blood supply may be partially or wholly cut off through an injury to the wall of the supplying artery, through thrombosis, embolism, arteriosclerosis, ligature, and pressure of tumors or of inflammatory infiltrations and exudates. The return flow may be obstructed, as from pressure, inflammation, or coagulation of the blood. The capillaries may be occluded from similar causes. Prolonged

stasis of blood will lead to death of the affected parts. A weakened heart action may be a factor of greater or lesser importance in some cases. Familiar instances of necrosis, due in the main to interference with the circulation, are: bed-sores; the local death which follows too tight bandaging, improperly applied splints, or the pressure of an elastic stocking; gangrene of the intestine from incarceration or from torsion of a part. In the cases of tumors the growth may progress far in excess of its nutritive supply, and gangrene will thereupon follow. To some extent mechanical influences play a part, as vessels may be compressed or twisted in the course of the growth of a tumor. This is seen, for instance, in the necrosis which occurs in the pedicles of pedunculated fibromata and lipomata.

Ligation of the principal arteries, when they are healthy and in healthy people, is attended by little danger of gangrene. The effect of ligation is to rupture the intima of the vessel and thereby to induce thrombosis. The circulation is quickly cut off, but usually sufficient time is given for the establishment of a collateral circulation.

Toxic agents of a great many kinds may bring about cell death, either by their direct deleterious action upon the cellular protoplasm or by inducing changes in the circulation. Such substances appear to enter into chemical union with the protoplasm of the cells or intercellular substance in such a way as to render life impossible. The most important are the various bacterial toxins, such as those of the staphylococcus, streptococcus, typhoid, diphtheria, tuberculosis, and cholera micro-organisms. Some few are derived from forms of animal life. Another class includes toxins which result from faulty metabolism within the body. Thus, uric acid, the biliary acids, the abnormal products occurring in diabetes mellitus, the pancreatic ferments, may, under certain circumstances, give rise to necrosis.

Inflammation is not infrequently accompanied by necrobiotic and necrotic changes in the tissues. This is due to a variety of factors. We have, for example, the effect of stasis, alterations in the composition of the blood, the toxic influences of substances derived from bacteria, and the pressure of inflammatory products.

Lastly, necrosis may, according to some authorities, originate in the inhibition of impulses from the central nervous system (neurotrophic necrosis). Probably in such cases the death of tissue results more from interference with the vascular mechanism than from simple cutting off of the trophic influences. Moreover, once the vitality of a structure is lowered, bacteria readily make their way into it and their influence must contribute to the final result.

The causes just mentioned may act separately, but not infrequently several are combined in a given case. The amount of necrosis resulting depends upon the nature and intensity of the operating cause, the length of time during which it is effective, and the vitality of the affected part. Tissues with weak resisting

power, such as are found in conditions of old age, general anæmia, cachexia, and marasmus, may undergo necrosis from a trifling cause.

The Forms of Necrosis.—The essential changes in the cell which indicate the presence of necrosis are destruction of the nucleus and more or less disintegration of the cytoplasm. These are present in every case. These changes may, however, be so modified by or associated with other processes that we are able to recognize different varieties according to the gross or the microscopical appearances presented. The form of necrosis depends upon the position and character of the affected cells, the nature and intensity of the causative agent, and the nature of the neighboring tissues. If the dead cells, for instance, are on the surface of the body, where evaporation can take place, the cells become inspissated and the part dry and mummified. If there be an abundant supply of fluid, the cells become hydropic and the part may liquefy. Again, the conditions may be favorable for the coagulation of lymph and the formation of fibrin, either in the cells or in the intercellular substance. Finally, the character of the necrosis may be modified by the occurrence of inflammation and the presence of putrefactive bacteria. The line of demarcation between these various forms cannot, however, be always closely drawn. One form frequently passes imperceptibly into another.

We may, however, recognize the following forms of necrosis, which are fairly well to be differentiated the one from the other:

(1) Simple necrosis. (2) Coagulation necrosis. (3) Colliquative necrosis. (4) Dry gangrene, or mummification. (5) Moist gangrene. (6) Caseation. (7) Fat necrosis.

Simple Necrosis.—In this form of necrosis the characteristic features are the disappearance of the nucleus, with hyaline or granular changes in the cellular protoplasm. The cells are often somewhat enlarged, but their general outline is well preserved. Occasionally, the cells seem hyaline and homogeneous. So far as gross appearances are concerned organs so affected are yellowish or grayish in color and diminished in consistency. The condition seems to be an advanced stage of cloudy swelling. It may affect any tissue, but is most commonly found in the specific epithelium of secreting organs. The liver and kidneys are very frequently attacked in cases of infection or intoxication. Thus, the cells lining the contorted tubules of the kidney often show marked necrosis in cases of mineral poisoning and in the cachexia of carcinoma. Large, irregular, necrotic areas of yellowish color are often met with in the liver in cases of appendicitis where infection has extended into the portal vein. The so-called "self-digestion" of the pancreas, described by Chiari, presents an accurate picture of simple necrosis, but is in most cases, if not in all, a post-mortem phenomenon. Gastric ulcers and the local necroses following severe burns are probably to be included in this category.

The so-called "focal" necroses demand a word or two. These are small local foci of cellular death found in lymph nodes and in the various parenchymatous

organs, the result of the presence in the blood of bacteria or their toxins. The condition was first observed by Oertel in diphtheria, but is met with in other affections, notably in typhoid, tuberculosis, and in the liver in puerperal eclampsia. It has been produced experimentally by the injection of the toxins of diphtheria, of ricin, abrin, and of vegetable toxalbumins. Capillary thrombosis may to some extent aid in the process, but the bacteria or their toxins are believed to be the chief causative factor.

Coagulation Necrosis.—There are several forms of necrosis which have considerable similarity, so far as superficial appearances are concerned. These are simple necrosis, coagulation necrosis, hyaline degeneration, and caseation. They all are characterized by destruction of the cells and the production of a hyaline or granular structureless detritus. By many simple necrosis is described as coagulation necrosis. It is perhaps better to reserve the latter term for that form of cell death in which there is a production of fibrin or fibrin-like material (fibrinoid degeneration). That there is such a form of necrosis may be readily demonstrated by the use of Weigert's fibrin stain. Coagulation necrosis occurs only in tissues rich in albuminous substances, and, theoretically, will result whenever, owing to the cell destruction, fibrin ferment is liberated to combine with the fibrinogen which is present in the lymph. The process is believed to be practically identical with that of coagulation of the blood and the formation of a thrombus.

Two forms may be distinguished—intercellular and intracellular. In the former, fibrin is formed between the dead and dying cells. This fibrin may be laid down in the form of threads, granules, or hyaline-looking masses, along with which may be recognized the débris of the original cells. The diphtheria membrane may be taken as the type of this form of necrosis, which affects most commonly mucous and serous surfaces (diphtheritic, croupous, or membranous necrosis). All forms of inflammation of mucous surfaces, associated with the formation of a membrane, are commonly referred to as diphtheritic, but this is somewhat confusing. It is better to restrict the term *diphtheritic* to *diphtheria*—that is to say, inflammation due to the Klebs-Loeffler bacillus—and to speak of the other forms as *diphtheroid.* Focal necroses in the internal viscera are not infrequently coagulation necroses, and there is also quite often a formation of fibrin in tubercles. Infarction less frequently gives rise to the production of fibrin. Superficial burns, if extensive, produce areas of coagulation necrosis in the spleen and lymph nodes.

The second variety of coagulation necrosis is characterized by the transformation of the parenchymatous cells of an organ or tissue into a solid or semisolid albuminous substance more or less like fibrin. As an instance of this we may take the so-called vitreous, waxy, or hyaline degeneration of striated muscle, known as Zenker's necrosis. This condition is found most often in prolonged fevers, such as typhoid, in some anæmic infarcts, in muscles which have been

subjected to heat or cold or to the influence of toxins, or which have been torn across. In the fevers the abdominal recti, the adductors of the femur, and the ilio-psoas are the parts most often affected.

Muscles so affected are semitranslucent and of a pearly-white or grayish color somewhat resembling raw fish. Microscopically, the muscle fibres are swollen, have lost their striations, and have a hyaline, homogeneous appearance. The exact nature of the process in these cases is somewhat doubtful. Friedreich, Weigert, and others, look upon it as a coagulation of the muscle plasma; others, as an inspissation of the albuminous constituents. In a few cases coagulation necrosis may result from the imbibition of fibrinogen-containing fluids and their subsequent coagulation within the cells.

Colliquative Necrosis.—In colliquative necrosis or liquefaction the dead material undergoes softening and to some extent solution. It occurs as a primary change or secondarily to some other form of necrosis. Colliquative necrosis usually occurs in tissues freely supplied with lymph and containing but little of the fibrin-forming substances. It therefore is found most often in the skin and central nervous system. Anæmic necrosis of the brain and cord is always associated with softening. The destroyed nerve-material is converted into a soft detritus, consisting of fragments of cell chromatin, droplets of myelin, and fat, which gradually become dissolved in the lymph. The necrotic material is often colored from the admixture of blood or blood pigment (red softening, yellow softening). In such cases absorption of the dead material may take place, with, if the area is small, the formation of a cicatrix. Larger patches of softening become enclosed in a fibrous capsule, and a certain amount of the detritus is absorbed, resulting in the production of a cyst filled with clear fluid. The softening that occurs in thrombi and in the walls of atheromatous vessels is an example of this form of necrosis. Liquefaction is also not uncommonly found in tumors. Primary colliquative necrosis is well seen in burns of the second degree, the first stage of vesication being an outpouring of lymph and a hydropic degeneration of the epithelial cells in the deeper layers of the skin.

Liquefaction necrosis may be secondary to simple or coagulation necrosis. Fibrinous exudates, as in pleurisies and in pneumonic lungs, in the later stages undergo softening, which is an important factor in the process of resolution. Areas of moist gangrene and caseation may undergo liquefaction. In abscesses liquefaction is a constant feature. Here, not only do we have the effects of the abundant outpouring of lymph, but we have the digestant action of ferments derived from infective bacteria. Conversely, coagulation may follow liquefaction, the fibrin-forming substances being derived from the leucocytes.

Histologically, areas of colliquative necrosis show, in addition to actual destruction of the cells, vacuolation, clear spaces between the cells, and a stringy detritus.

Caseation.—Caseation is a term applied more or less loosely to designate that

form of death of tissue which is characterized by the production of material somewhat resembling cheese. It is to be regarded probably as a post-necrotic change rather than as a form of necrosis. Simple and coagulation necrosis and moist gangrene may be followed by caseation. Caseation is found typically in certain of the infectious granulomata, notably tuberculosis. Somewhat similar changes occur in gummata and in actinomycosis, and occasionally in tumors. Caseous foci are opaque, grayish-white or yellow, more or less firm and granular, and are cheesy in consistence. If they are hard and dry, we speak of the process as firm or hard caseation; if the imbibition of fluid has occurred, we speak of it as soft caseation.

Microscopically, we find more or less extensive areas in which the normal out-

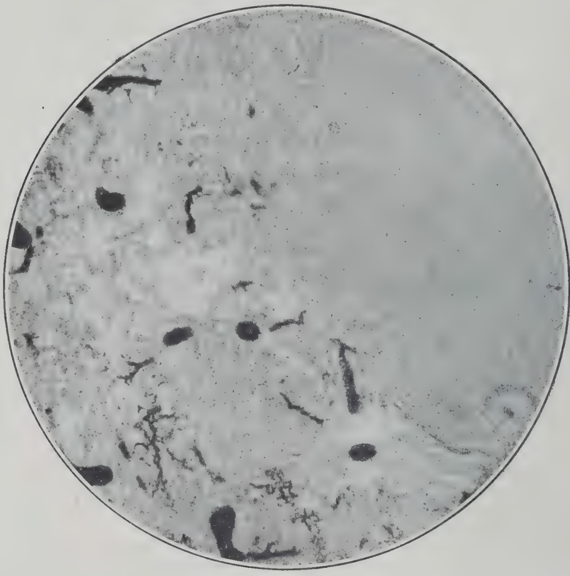

Fig. 70.—Caseation (Tuberculous) in the Lung. (*Leitz obj. No.* 3.) Area of caseation to the right; the blood-vessels injected to show the avascularity of the necrotic part. (*From the author's private collection.*)

lines of the cells and tissues are lost, the cells in various stages of disintegration, with liberation of their nuclear chromatin. In the larger areas the central portion is converted into a structureless, granular mass, consisting of cellular débris, fat, and sometimes calcareous salts. In some cases fibrin is present. In the case of tuberculous caseation, it is believed that the necrotic change is due in part to obstruction of the nutrient blood-vessels, and in part to the influence of the toxins produced by the bacilli (see Fig. 70). Somewhat similar caseation is occasionally observed in certain non-tuberculous inflammatory exudates in the lungs.

Caseous foci may be completely absorbed and cicatrized. They may soften or become calcified. When they tend to heal they become in time surrounded by a fibrous capsule.

Fat Necrosis.—This is a curious form of necrosis of considerable interest to

the surgeon. In the vast majority of cases fat necrosis is associated with some lesion of the pancreas, such as pancreatitis, although Fitz holds that it may occur in the absence of pancreatic disease. Experimentally it has been produced by the injection of pancreatic extract into adipose tissue, the introduction of certain substances into the pancreatic duct, ligation of the pancreatic vessels, the introduction of pieces of pancreas into fatty tissues or into the peritoneal cavity, and by the action of steapsin upon fat.

The necrotic areas vary in size from that of a pinhead to a pea, are opaque, grayish, yellowish, or sometimes black in appearance. They are usually sharply defined, and on section are soft or gritty. Such areas may be found in the pancreas, in the peripancreatic fat, in the omentum, and also occasionally in the fat of more distant regions, such as the pericardium, liver, bone-marrow, and retroperitoneal tissues. In some cases the pancreas itself may be free.

Microscopically, the parts so affected show that the fat cells are enlarged and the nuclei absent. The cellular substance is granular or presents the appearance of fine needles radiating from the centre. Osmic acid does not stain the necrotic material, while it tinges the healthy fat black or brown.

The areas of fat necrosis may liquefy or become calcified. The condition usually ends in the death of the patient, but it may be recovered from. Extensive fat necrosis may be associated with diffuse hemorrhage into the pancreas or with sequestration of large portions of this organ.

The researches of Hildebrand and Flexner have shown that fat necrosis is due to the liberation of the fat-splitting ferment of the pancreas. This acts upon the neighboring fat to produce fatty acids, which ultimately unite with the calcium salts.

Gangrene.—Gangrene, or necrosis of the soft parts, is a term somewhat loosely employed by surgeons to designate certain peculiar changes which occur in dead tissues. The leading features of gangrene are that the tissues die in bulk, and that this death is accompanied by putrefactive changes in the affected area. A number of other terms are used also at times to express more or less completely the same underlying idea—*mortification, putrefaction, putrescence, sphacelation.*

Gangrene may be *primary* or *secondary.* In primary gangrene the condition is due to the direct action on the tissues of a micro-organism having certain peculiar powers, and is to be regarded as a specific infection. In secondary gangrene the necrosis is due to some other cause, and the affected part is subsequently invaded by putrefactive bacteria.

The etiological factors at work in the causation of gangrene are somewhat varied. The most important single cause is obstruction to the arterial blood supply of a part. Traumatism, or certain toxic agents, or, again, bacteria, may lead to death of a part by direct local action. Other cases are neuropathic in origin.

Primary gangrene includes a number of specific affections, such as infection

with the B. Welchii, B. œdematis maligni, B. diphtheriæ, B. anthracis, B. coli, and some other imperfectly known organisms. Under certain circumstances these germs have been known to set up severe local inflammation followed by gangrene of the part. They appear to be competent to produce gangrene by their unaided action, but in some cases there may be a combined or secondary infection with putrefactive micro-organisms. On occasion, they may be implicated in the causation of secondary gangrene.

Fig. 71.—Dry Gangrene of the Foot.

Secondary gangrene is much the more common variety. The original necrosis may be due to vascular disturbances, alterations in the composition of the blood, pressure, the influence of thermal, chemical, or physical agents, infection, or neuropathic disturbances, the process being characteristically modified by the subsequent entry of parasitic and saprophytic micro-organisms.

Anatomically we may recognize two main varieties of gangrene—*dry gangrene* or *mummification,* and *moist gangrene.* Both forms are essentially the same, any differences being due to varying physical conditions. Both are forms of necrosis and both are accompanied by putrefactive changes.

Many different forms of gangrene are described. They may be classified according to etiology, according to their clinical course, or according to their distribution.

According to causes of origin we have: (1) Gangrene from vascular obstruction, (2) traumatic gangrene, (3) inflammatory and infective gangrene, (4) neuropathic gangrene, (5) "idiopathic" gangrene.

According to the clinical course we may recognize: dry gangrene (Fig. 71), moist gangrene, emphysematous gangrene, putrid gangrene, circumscribed, diffuse, spreading, or phagedenic gangrene.

According to distribution may be differentiated: localized (Fig. 72), multiple, metastatic, symmetrical gangrene.

Most writers on systematic surgery do not adhere exclusively to any of these modes of classification, but describe the most striking clinical types on their merits without much reference to the above considerations. It is better, however, to have some logical method.

Among the different forms of gangrene there is only one concerning which we shall take the liberty of making a few remarks—viz., noma. Presumably all the different forms will receive full consideration in the article on Gangrene which is to appear in a later volume.

Noma.—Noma (cancer aquaticus, Wasserkrebs) is a particularly rapid and fatal form of gangrene, which usually attacks the face or the pudenda. It is found without exception in debilitated or cachectic children, generally between the ages of two and twelve, and usually attacks those who are already suffering from one of the acute infective fevers. Rarely, it may arise independently, or as a sequel of acute ulcerative stomatitis. Noma of the face begins usually in the buccal mucous membrane near the angle of the mouth, occasionally in the gums. The affection first makes its appearance as a livid, swollen patch. Small vesicles form and the tissues present a grayish-yellow inflammatory infiltration, which rapidly breaks down and becomes gangrenous. The process quickly spreads to the skin of the cheek, so that the whole thickness of the cheek is converted into a blackish, necrotic substance, about which the tissues are markedly infiltrated and œdematous. (See Fig. 73.) No proper line of demarcation, in the ordinary acceptation of the term, is formed. The gangrene is usually unilateral, but may extend to the opposite side and even attack the bones of the nose and jaw.

Fig. 72.—Gangrene of the Appendix Vermformis in Acute Appendicitis ; Concretion. (*Pathological Museum, McGill University.*)

At the vulva the process usually commences at the margin of the labia, and may eventually spread to the clitoris, nymphæ, hymen, and urethra. It may even invade the perineum, anus, thigh, and mons veneris, and, like noma of the mouth, seems to have a tendency to penetrate deeply and attack the bone.

Noma may be attended with high fever, chills, and great prostration, but the special symptoms are not infrequently masked by those of the previously existing disease. The condition is exceedingly fatal, and the patient usually sinks into a state of profound prostration and rapidly succumbs. Noma of the mouth is said to be occasionally complicated by gangrene of the lungs and enterocolitis.

The affection appears to be almost certainly of infectious nature. It not infrequently occurs in epidemics and affects parts that are particularly exposed to the action of micro-organisms. The specific cause, if there be one, has not been demonstrated as yet. A bacillus, resembling that of diphtheria, has been described by Bishop and Ryan and by Schimmelbusch, but is not invariably present. Babès and Zambilovici have isolated from some cases a pathogenic microorganism capable of inducing gangrene when injected into rabbits. Ranki and Lingard have also described a germ which they believe to be specific.

FIG. 73.—Noma or Cancrum Oris. (*Case of Dr. A. T. Bazin.*)

Necrosis of Bone. — Death of bone occurs under two forms—*necrosis* and *caries*. Necrosis is death of bone *en masse*, and is analogous to gangrene of the soft tissues; caries is a gradual and almost imperceptible disintegration of bone into fine particles, which may be compared to ulceration.

Necrosis of bone may be due to inflammation, traumatism, interference with the circulation, thermal or chemical agents. Occasionally the death of the bone substance is direct, but in most cases there is obstruction to the afferent blood supply, however it may be produced. To this we must add in some cases the disintegrating action of bacterial toxins.

Necrosis of bone can, in rare instances, be attributed to embolism. The circulation is under normal circumstances fairly active, and should a nutrient artery become blocked, a collateral circulation is readily established. In the few instances where necrosis has followed embolism, the smaller arterioles and capillaries have been obstructed. We occasionally see, in cases of tuberculosis of the long bones, wedge-shaped areas at the ends, having the base of the wedge directed toward the articular surface. This suggests infarction, a view which is strongly corroborated by the experiments of Mueller. It has been shown, too, that the articular surfaces are supplied by terminal arteries, so that the possibility of the occurrence cannot be denied. Volkmann met with an instance of multiple necrosis of the tibia and astragalus in mitral endocarditis. In such a case we probably have to do with multiple capillary emboli. These emboli may be simple or infective. Simple necrosis or an abscess may thus result, or what was at first a simple necrosis may be converted into one of a suppurative or tuberculous nature.

Bones receive their nourishment through numerous freely anastomosing vessels situated in the marrow and periosteum. These are connected with small vessels in the Haversian canals, so that an abundant supply of blood is furnished to every part of the bone. Anything, then, which damages the medulla or periosteum, or which obstructs the circulation within the bone itself, may give rise to necrosis. Mere separation of the periosteum does not appear to be competent to produce necrosis, but if the surface of the bone be laid open to the external air, or if there be a suppurative process which has extended to the Haversian canals, necrosis will follow. Necrosis, then, may result from suppurative periostitis, osteitis, or osteomyelitis, or from analogous tuberculous or syphilitic lesions. All these conditions, it will be observed, result in compression of the blood-vessels from inflammatory exudates, and may lead to thrombosis or to endarteritis. Suppuration or ulceration of adjacent parts may also extend to the periosteum, and so give rise to necrosis of the bone.

Traumatism may produce necrosis, provided that it be of such a nature as to cause the separation of portions of the bone from their natural attachments. If a bone be splintered, the minuter fragments may in time be absorbed. Larger ones may become reunited, provided that the wound remain aseptic, as has been shown experimentally by Ollier, Bergmann, and others. This has an important bearing on the surgical procedure of transplantation of bone. Experiments have proved that detached pieces of bone may be successfully transplanted from one part to another, and even from one animal to another, if suppuration do not take place. This does not invariably hold good, for Winiwarter observed total necrosis of the bone to take place in two cases of subcutaneous dislocation of the astragalus, in spite of careful reposition of the parts. Where the bone is extensively crushed, vessels are lacerated or compressed by portions of misplaced bone or blood-clot, and necrosis therefore readily takes place.

A good example of necrosis resulting from toxic or chemical agents is the phosphorus necrosis. This is met with in people employed in the manufacture of phosphorus matches, and is due to the injurious action of the phosphorous vapor. Phosphorus necrosis is not so common as it used to be, owing to the more extensive introduction of other kinds of matches and the stricter enforcement of hygienic measures.

Phosphorus necrosis affects usually the lower jaw, less often the upper. Lack of attention to the cleanliness of the mouth, and the presence of carious teeth, predispose to the condition. The disease usually begins, as Wegner has shown, with inflammation of the periosteum, which, under the stimulating influence of the phosphorus, takes the form of a hypertrophic or productive periostitis. Subsequently, owing to the action of micro-organisms, infection takes place with the production of suppuration and secondary necrosis between the periosteum and the new bone or between the new and the old bone. Rarely, the

disease begins more acutely without the preliminary hyperostosis. In time the whole of the lower jaw may become necrotic.

The Mechanism of Bone Necrosis.—When a portion of a bone dies, it is gradually separated from the living tissues and may in time be completely separated or exfoliated. This process is called *sequestration*, and the separated bone a *sequestrum.* Sequestration results from a process known as lacunar resorption. The ordinary breaking down of bony substance takes place through the agency of certain large cells called osteoclasts (myeloplaxes). These are situated in the bone marrow and the deeper layers of the periosteum, and erode their way into the bone, giving rise to minute excavations, known as Howship's lacunæ. In the pathological conditions of bone under consideration, these osteoclasts are greatly increased in numbers and lie closely packed together. Therefore, rarefying osteitis, as it is called, leads to rapid destruction of the dead material, and may succeed, in the case of the smaller fragments, in completely removing it. The process of lacunar resorption begins at the line between the living and the dead material, and results in the formation of a line of demarcation. The periosteal surface of the sequestrum often remains smooth, while the margins are rough and uneven. The process proceeds centripetally, resulting in the loosening of the fragment and a more or less marked diminution in its size. Inasmuch as the dead bone is to all intents and purposes a foreign substance, there is a certain amount of reactive inflammation in the neighborhood which tends to hasten the process. If, as is so often the case, the necrosis be due to inflammation, we may have the formation of pus, which accumulates in and about the sequestrum and in time makes its way to the surface of the body. In this way communication is established between the site of the necrotic process and the external air (sinus, fistula, cloaca). Through such fistulæ portions of the dead bone, if lying free upon the surface of the bone, may make their way to the exterior and be cast off, and healing will in many cases result. Sequestra, however, which lie in the interior of the bone, if not absorbed, remain incarcerated unless removed by operation.

Coincidently with the separation and removal of the dead material, in cases where repair is possible, there is an attempt at the restoration of the damaged part through reactive bone formation. The osteophytes of the periosteum are stimulated to increased activity, so that a capsule of newly formed bone is produced around the sequestrum (involucrum). This is particularly well seen in cases of total destruction of the shaft of a long bone, where a complete new diaphysis may be developed from the inner layer of the periosteum, which gradually restores the continuity and configuration of the bone to such a degree that eventually no deviation from the normal can be detected. This formation of new bone is most marked in the case of young and vigorous subjects. Where a suppurative inflammation is going on, pus may escape from the cavity through the fistulæ. If it be pent up, however, it may in its turn lead to further necrosis, even of the

newly formed bone. In long-standing cases, where the power of repair is very marked, the inflamed bone in the neighborhood of the necrotic part becomes hard and eburnated (sclerosing osteitis).

Sequestra may be divided, according to their situation, into external, or peripheral, and central. The variety termed by Blasius "necrosis tubulata" is very rare. The chief characteristic is a tubular sequestrum, the internal axis of which is formed of living bone connected with the old bone.

Dead bone is dry, light, devoid of fat, and of a whitish color, owing to anæmia. It may be porous or, on the other hand, sclerosed.

ULCERATION AND CARIES.

Closely allied to the conditions we have been describing, and, from the pathologist's standpoint, practically identical with them, are *ulceration* and *caries.* The terms gangrene and necrosis connote, as we have pointed out above, the idea of death of tissue in bulk; ulceration and caries, as these names are ordinarily employed, signify death by the more gradual process of molecular disintegration. Authorities differ radically in their ideas as to what constitutes an ulcer. Billroth defines it as "a loss of substance, with no tendency to heal." Golding Bird holds it to be "a limited area of granulation tissue on the surface of the body." To my mind both definitions are defective, in that they are not sufficiently comprehensive, while to a great extent they are mutually exclusive. An ulcer is none the less an ulcer because it is healing, and, in fact, we are constantly hearing the term "healing ulcer." The second definition is too restricted, inasmuch as it excludes all ulcers which are not granulating, such as phagedenic and the so-called "croupous" ulcers. Pathologically speaking, we cannot very well separate the condition known as a granulating wound from a healing ulcer, but in the surgeon's mind there is nevertheless an underlying thought which distinguishes ulcers from all other superficial losses of substance. This appears to be the idea of erosion. Perhaps we can come very near to what is usually meant by the term "ulcer," if we define it to be *a superficial loss of substance of the skin or a mucous membrane, which at some or other has shown a tendency to enlarge its boundaries.* This definition would include those cases excluded by the other definitions referred to, and would exclude healthy granulating wounds resulting from traumatism, while it is at the same time non-committal on the question of etiology.

Ulceration is the process by which ulcers are produced.

Etiology.—Ulcers may be brought about by a great many different factors, which may operate singly or in combination. We may consider the subject conveniently under the headings of (1) predisposing causes and (2) exciting causes.

Predisposing causes are general or local.

General Predisposing Causes.—Age is of no great importance in determining

the frequency of ulceration. One might expect that ulcers would be relatively more common in those past middle life, considering the prevalence of retrogressive changes in such persons; but this, in a sense physiological, tendency to ulceration in the aged is more than counterbalanced by the frequency of tuberculosis and syphilis in the young and middle-aged, and of traumatism in the active period of life. Ulcers are said to be three times more prevalent in men than in women. This is probably due to the greater liability to traumatism in the case of males, lack of cleanliness, and the ravages of alcohol and syphilis. All occupations which expose to trauma and promote dirt would, of course, predispose.

Many constitutional diseases and those which lower vitality tend to invite ulceration. Diabetes, gout, anæmia, scurvy, tuberculosis, and syphilis are important in this connection. General obesity and disturbances of the cardiovascular system have also to be mentioned.

Local Predisposing Causes.—Of these, perhaps the most important is impaired circulation. Atheroma of the arteries, embolism or thrombosis in the arteries, veins, or capillaries, often lead to ulceration by cutting off the nutrition of the part. Small areas of ischæmic necrosis may be converted into ulcers. Stasis in the venous circulation, especially if accompanied by œdema, is a potent factor in bringing about ulceration. Thus, ulcers form on the extremities in obstructive valvular disease of the heart and varicose veins. In what way ulcers of the lower part of the leg should be connected with varicose veins has been a matter of debate. Varicose veins do not inevitably lead to ulceration, so that some other factor must play a part. Some find the connecting link in gout; others in a local neuritis. Probably it is more correct to find it in the phlebitis and periphlebitis which so often come on in the case of varicose veins, while from the superficial position of the lesion infection from the skin is readily brought about. Rupture of a vein with extravasation of blood into the surrounding tissues might cause ulceration in one whose tissues possessed a low resisting power. Generally, too, these conditions occur in the obese, in whom the circulation is feeble.

Obstruction to the lymphatic circulation may also lead to ulceration. This is seen occasionally in cicatricial closure of the lymphatics of a part after operations and in elephantiasis.

Trophic disturbances in the central or peripheral nervous system may also be provocative of ulceration.

Exciting Causes. — These are direct and local in their character. Chief among them should be mentioned traumatism of all kinds, various forms of infection, caustics and other toxic substances, and malignant disease.

Traumatism.—One of the most common direct causes of ulceration is injury in some form or other. The effect of an injury will, of course, depend upon the nature of that injury and the character of the part affected. Thus the skin or a

mucous membrane may be destroyed by a contusion, laceration, the operative removal of substance, by friction or by a burn. In many cases healing will begin immediately and will progress by the formation of healthy granulations. If, however, infection should be superadded, or should it have existed from the beginning, the wound may take on unhealthy action and tend to spread by molecular disintegration. On the other hand, a much slighter injury, occurring in a person of lowered vitality or in one the subject of constitutional disease or poor circulation, or with deranged nervous mechanism, may be followed by ulceration.

Besides sudden losses of substances mechanically produced, we have to mention ulceration resulting from pressure, extreme heat or cold, x-rays and caustics.

Pressure, either from within or from without, if continued for a length of time, may produce ulceration, partly by the direct action on the cells and partly by cutting off the nutrition. Bed-sores, ulcers from improperly applied splints or orthopedic apparatus are well-known instances of this. Pessaries or other foreign bodies in the vagina, calculi in the urinary bladder or the biliary passages, impacted fæces and fecal concretions in the appendix and bowel, hard substances introduced into the nasal passages, not infrequently cause ulceration. Deposits of various salts beneath the skin, as in cases of gout, tumors growing from below into the skin or a mucous membrane, the filaria medinensis or guinea-worm, lead to ulceration of the skin. In badly-performed amputations the flaps may ulcerate from pressure of the end of the bone or from too tightly drawn sutures.

Caustic substances, such as acids, alkalies, and certain acid salts, act by directly killing the part to which they are applied. Unless their effects are quickly neutralized, the cells for a considerable distance outside of the direct field of action may suffer in vitality and subsequently die, thus leading to a spreading ulcer.

Infection.—Theoretically it is possible to conceive of ulceration in the absence of infection. Practically, however, since ulcers are found in the skin and mucous membranes—in other words, on the surfaces of the body which are exposed to the attacks of micro-organisms—ulceration and infection always go together. The infecting agents act by converting what would otherwise be a granulating or healing lesion into one of a disintegrating and destructive character. The germs at work are usually the ordinary pyogenic or saprophytic organisms. In an analysis of one hundred cases of ulceration of the leg, Bukovsky found the B. pyocyaneus most frequently present. Other germs were staphylococci, streptococci, B. coli, B. proteus, and B. Friedländeri (one case), besides a few other relatively unimportant forms. Of course there are several forms of ulceration due to specific micro-organisms,—forms which are not represented in this analysis, and which usually receive separate consideration, such as syphilis, tuberculosis, actinomycosis, Madura foot disease, glanders, leprosy. Rapidly spreading ulcers, termed *phagedænic,* are due to a particularly virulent infection

in the case of a debilitated subject. Of this type are the hospital gangrene and the phagedæna which attacks venereal sores.

We have to bear in mind that infection may be the primary cause of ulceration, as in the specific diseases just referred to, in typhoid, and in chancroid, but not infrequently it is superadded to necrosis originating in another way. Thus traumatic losses of substance may become secondarily infected. Certain skin diseases, such as herpes, eczema, ecthyma, and pemphigus, if situated in parts of the body which are subjected to friction and imperfectly cleansed, often lead to infection and intractable ulceration. The eczema that so often accompanies varicose veins is an instance of this.

Ulceration may also be produced on mucous surfaces by the direct action of organisms like the B. typhi, B. dysenteriæ of Shiga, B. diphtheriæ, Amœba coli, and the plasmodium of malaria.

Parenchymatous inflammations, when not progressing satisfactorily toward healing, may result in ulceration. A good instance of this is seen in the somewhat common event of an abscess making its way to the surface of the body, or "pointing," as it is called. An abscess is a deep-seated focus of suppurative inflammation. The tissues disintegrate and there is formed a cavity filled with pus. This tends to increase in size in the direction of least resistance. The effect of the pus is to produce, first, pressure, then distention and stretching, and, eventually, molecular disintegration of the structures which bar its way. Finally, in favorable cases, the pus reaches the surface of the body or is discharged into some hollow viscus. In this way healing is not infrequently accomplished. The process in question may properly be regarded as an ulcerative one, inasmuch as there is a molecular death of tissue which tends to spread. In some cases the nature of the infection is such that healing does not take place, except by the aid of art, or else the whole track of the suppurative process becomes specifically infected and a more or less permanent external ulceration results.

Syphilis in all its stages is a potent cause of ulceration. The chancre is usually ulcerative in character and is an example of ulceration from a primary infection. In the secondary stage mucous patches and the various cutaneous lesions may in weakly and uncleanly individuals be converted into ulcers. The most typical syphilitic ulcer is, however, found in the tertiary period in the breaking-down gumma.

Tuberculous ulceration is found both in the skin and in the mucous membranes. Thus, we may have primary infection of the tongue, fauces, larynx, bronchi, stomach (rarely), and intestines. The skin also may be directly invaded by the tubercle bacilli. Again, both skin and mucous surfaces may be infected secondarily through the blood, or a tuberculous abscess may extend to the surface and there discharge, forming a more or less intractable ulcer. Tuberculous ulcers are usually indolent, with irregular, thickened edges and uneven base. The discharge consists of caseous detritus and, usually, pus.

In the case of *glanders*, soft nodules form in the mucous membranes, such as that of the nose, or under the skin, and these nodules break down, forming irregular ulcers which discharge a glairy pus.

The *actinomyces bovis* and *actinomyces* of *Madura foot disease* give rise to similar lesions, inflammatory granulomata, which, owing to secondary infection, soften and suppurate, and when near the surface often discharge externally, forming ulcers.

The ulceration so characteristic of *leprosy* is due either to breaking down of lepra nodules, to neurotrophic disturbances, or to the anæsthesia produced, which renders it impossible for the patient to perceive and avoid injury.

Toxic Ulceration.—Certain drugs in the course of their elimination through the emunctories may set up inflammation and, finally, ulceration. Such are mercury, which produces ulcerative stomatitis, gingivitis and colitis; and phosphorus, which causes ulceration of the buccal mucous membrane.

Ulceration in Tumors.—Benign tumors may undergo necrosis and ulceration. This occurs when the tumor is so large that its nutrition is impaired, or when its pedicle is kinked or twisted, thus interfering with the blood supply. Large pedunculated fibromata and lipomata not infrequently undergo ulceration. In the case of the lipomata, owing to the liberation of fatty acids, very foul ulcers are produced.

Malignant tumors, carcinomata and sarcomata, regularly break down, and, if on the surface of the body, ulcerate after they have attained a certain size. Good examples of this are found in the epitheliomata of the skin and mucous membranes, rodent ulcers, chancroids, and melanotic sarcomata.

Secondary malignant growths may extend from the deeper parts to the skin and mucous surfaces, and then undergo necrosis.

The Locality and Distribution of Ulcers.—In general, ulcers are apt to be found in those parts of the body which are exposed to injury or infection, and in which the circulation is poor. Therefore we find them on the uncovered portions of the body, at the orifices, in the mucous membrane of the alimentary tract, and on the extremities. Those due to metastatic infection, carcinosis, or sarcomatosis, are usually multiple, and may, of course, develop anywhere. Ulcers are, therefore, common on the cornea, at the junction of the skin and mucous membranes, as, for example, at the angles of the mouth and at the anus, on the nipples, in the stomach, intestines, the urinary and biliary passages.

The Pathology of Ulceration.—The pathological process at work in ulceration is in the main the same in all cases, although it differs in minor details according to the causative factor. There are two opposing forces in operation: First, disintegration of tissue; and secondly, in most cases a more or less perfect attempt at repair, which manifests itself after a variable period. All cases are accompanied by the phenomena of inflammation. We may recognize two great classes of ulcers—one in which the destruction of tissue is the direct result of cir-

culatory disturbance or trauma, to which an inflammatory process is subsequently superadded; the other in which inflammation is the primary cause of the cellular disintegration.

The first class of cases, which are pathologically and etiologically related to atrophy and degeneration, is represented by the ulceration which is produced by ischæmic necrosis and gangrene, passive congestion and œdema of tissues, contusions, the pressure of tumors or surgical appliances.

The second class of cases includes such conditions as the ulceration which results from infected wounds, diphtheritic inflammation, and the specific granulomata.

Owing to the great variety of the causes that produce ulceration and the differences in the local reaction, it is impossible to give one description which will apply to all cases. It will therefore be better to indicate the chief types. We may classify ulcers according to their etiology or according to their appearance and clinical course.

According to etiology we can recognize traumatic ulcers, ulcers from stasis, inflammatory, gouty, scorbutic, neuropathic, and malignant ulcers.

It is perhaps more usual in practical works on surgery to classify ulcers according to their clinical features. Thus we have the *simple*, healthy, or healing ulcer, the *weak* or œdematous ulcer, the *inflamed* ulcer, the chronic callous or *indolent* ulcer, the *fungous* ulcer, the *irritable* or painful ulcer, the sloughing or *phagedænic* ulcer, *varicose*, *eczematous*, *gouty*, *scorbutic*, *specific*, and *malignant* ulcers. These terms indicate in part the particular causes of the ulceration, and in part their special characteristics, due to local conditions and surroundings. The local conditions are, however, liable to change from day to day, so that various gradations occur between the various forms of ulcers, and even the type itself may change from time to time. For example, a callous ulcer may be transformed into a phagedænic one.

The Simple Ulcer.—This may be taken as the type of all ulceration. Others differ from it merely in detail, and all ulcers tend when healing to approach this form. The base of the ulcer is level or nearly so, and covered with healthy granulations. The edges are smooth and shelving. The newly formed epidermis, destined to cover the damaged area, can be seen at the borders as a thin, bluish-white film. The discharge is creamy, inodorous pus, or possibly, if the ulcer be kept clean and dressed antiseptically, serum.

Microscopically, the base of the ulcer consists in the main of inflammatory round cells, together with some spheroidal and epithelioid cells. On the surface may be pus cells, fibrin, cell débris, and dried serum. Deeper down we find greater amounts of connective tissue, with, if the ulcer be healing satisfactorily, young fibroblasts. Newly-formed capillaries are also present, in the form of vertical sprouts and loops extending in the direction of the surface. Round about the ulcer will be found a variable amount of connective tissue or scar tissue,

which contracts as the ulcer heals. At the margin the epithelium is proliferating by division of the cells. In the case of the skin proper, the papillæ are not reproduced nor are the hair follicles and various glands.

As the process of healing progresses, more of the round cells are produced than necessary, and are cast off in the discharge. Loops of blood-vessels are abundantly produced and carry along with them numerous fibroblasts, which are to be converted into cicatricial tissue. Fibrous tissue is produced in increasing amount, and the epithelium gradually extends over the raw surface until the loss of substance is made good. Finally, many of the blood-vessels disappear, and the scar contracts, becoming firm, pale, and anæmic.

The Weak or Œdematous Ulcer.—Any ulcer may become weak if healing be too long delayed. This form is generally found in connection with tuberculous bones and joints. The edges and tissues about the ulcer are generally healthy, but the granulations on the surface are abundant, flabby, semitranslucent, œdematous, and friable. The discharge is watery and free.

The Fungous or Exuberant Ulcer.—Here the edges are healthy, but the granulations rise above the surface, are tumid, dark red, redundant, and easily bleed. The condition is usually due to some obstruction in the return venous circulation.

The Inflamed and Inflammatory Ulcer.—In these ulcers inflammation is the most striking feature. The inflammation may result from some constitutional vitium, as from alcoholism, improper and insufficient food and other causes of impaired nutrition, or from any local cause of irritation. Inflammatory ulcers are irregular in shape; the edges are ragged and shreddy or sharply defined. The base is dry, dull red, devoid of granulations, covered with serous or sanious discharge, sometimes with yellowish sloughs. The surrounding tissues are swollen, red, and hot.

The Sloughing or Phagedænic Ulcer.—This is a more intense form of the inflammatory ulcer. The destructive processes are greatly in the ascendant, and the inflammation is of quickly spreading and infective character. The base is devoid of granulations, secretes an ichorous discharge, and is converted into an ashen-gray or black, sloughy material. The edges are irregular, swollen, and undermined. The process appears to be due to a specific micro-organism, as the ulceration proceeds with extraordinary rapidity unless checked by appropriate measures. There is usually also considerable constitutional disturbance. This form of ulceration is seldom seen except in connection with venereal disease in those with broken-down constitutions.

The Chronic, Callous, or Indolent Ulcer.—An ulcer may become indolent as a result of long-continued irritation. The edges of such an ulcer are smooth, white, hard, rounded, and insensitive, and they are indurated from the presence of inflammatory products, so that the circulation is impaired and healing prevented. The neighboring tissues are congested, and the skin is often excori-

ated or eczematous. Granulations are either absent or are scanty, small, and badly formed. The discharge is thin and sanious. Indolent ulcers are commonly found in the lower third of the leg, may exist for years, and are attended by but little pain. They may be small or may gradually extend round the limb. Sometimes they become adherent to the fascia, periosteum, or bone. When very old or when subjected to constant irritation, they may take on epitheliomatous action.

The Irritable or Painful Ulcer.—Any ulcer may be irritable and painful or may become so, but the term "irritable" is generally restricted by surgeons to painful fissures about the anus and to a small, superficial, congested ulcer, found usually in women after middle age, near the ankle. The pain is extreme and is believed to be due to involvement of the nerve endings.

Varicose and Eczematous Ulcers.—This form of ulceration, as the names imply, is associated with varicose veins and eczema. Both conditions are not infrequently found together.

Gouty Ulcers.—Gouty ulcers are found over uratic deposits. They are small and superficial, and the discharge contains sodium urate, which it deposits as a chalk-like material about the ulcer.

The Scorbutic Ulcer.—The surface of a scorbutic ulcer is covered with a spongy, dark, adherent, fetid crust. When this is removed the surface bleeds freely and the same material is reproduced.

Specific Ulcers.—These include tuberculous and syphilitic ulcers.

The former are generally multiple and often confluent. In the neighborhood can sometimes be found traces of former ulcers in the form of scars and depressions. Tuberculous ulcers are generally due to the breaking down of tuberculous nodes, with discharge of their contents externally. The granulations are pale, œdematous, protruding, and bleed freely when touched. The discharge is scanty, thin, and yellowish-green in color. The edges are pale, thin, and undermined. *Lupus*, or tuberculosis of the skin, is described elsewhere.

Syphilitic ulcers, with the exception of the primary sore, are divided into *superficial* and *deep*. The superficial are usually associated with syphilitic eruptions. They are circular or crescentic in shape, or, when several have coalesced, serpiginous or annular. They spread by their convex aspect, while the older portions tend to heal. The base of such ulcers is but slightly depressed, of dark reddish color, and is covered with a scab or slough. The edges are sharply cut and surrounded by a dull reddish areola.

Deep syphilitic ulcers are due to the breaking down of gummata. They are oval or circular in shape. The base is depressed and covered with a yellowish slough resembling wet wash-leather. The edges are steep, well-defined, slightly excavated, and of a dull reddish appearance.

Malignant Ulcers.—These include epithelioma, carcinoma, sarcoma, and rodent ulcers. They are described elsewhere. In general it may be said, how-

ever, that benign growths may produce ulceration from pressure on the skin arising in the course of their growth, or from impairment of the circulation within them. In the case of malignant tumors, when ulceration occurs, it is due to the breaking down of the cells proper of the growth. The surrounding tissues may in time be invaded by the new growth, and this freshly formed material may in its turn break down. Usually in such cases there is more or less inflammation, with its ordinary phenomena superadded. Both primary and secondary new growths may attack the skin and mucous membranes and undergo necrosis. Chronic ulcers in elderly people and lupous patches may at times undergo epitheliomatous transformation.

There are a few forms of tropical ulcers, mostly of uncertain etiology, about which a word or two may not be out of place here.

Veldt sores are a form of ulcer common among the British troops during the recent war in South Africa. The sores are found on the exposed parts of the body, the hands, forearms, and feet. They appear to begin in the deeper layers of the epidermis, and at first resemble a bleb abrasion. Later, a slowly spreading, chronic ulcer is formed. The etiology is still under discussion.

Delhi sore is met with in India, Central Asia, the Levant, Algeria, and the Malay peninsula. It is found on some exposed portion of the body, and begins as one or more papules, which become pustular and finally develop into ulcers. The ulcers may be multiple and may fuse together. The base is usually irregular, healing in one place and spreading in another. The edge is thickened, ragged, and surrounded by an areola of inflammation. The ulcer runs a very sluggish course. When it heals it leaves a depressed scar, puckered in the centre, and of a bluish-brown color.

Annam ulcer is a variety of phagedæna found in Annam, Aden, Cochin China, and Mozambique. It usually begins on the foot or leg as an area of infection. This sloughs and a more or less rapidly spreading ulcer is produced. The base is covered with unhealthy granulations, which bleed at the slightest touch, or with a grayish pseudo-membrane, and discharges fetid pus. The edges are undermined. Both base and edges may be extensively gangrenous, and the ulcer may penetrate deeply. The cause is unknown, but syphilis, anæmia, bad hygienic conditions, are believed to predispose.

Gaboon ulcer is found in natives of the Gaboon. It occurs on the limbs and is similar to a syphilitic ulcer.

Dracuncular ulcer is endemic in parts of India, Arabia, Bokhara, Turkestan, tropical Africa, and South America. It is due to the Filaria medinensis, or Guinea-worm, a species of thread-worm. This is a very large filaria, averaging three feet in length, but may be as much as six feet long. It lives in the subcutaneous tissues. The female worm, which is the one that causes the trouble, as she approaches maturity, works her way to the surface, usually in the leg, foot, or ankle. She then discharges her eggs and penetrates the skin, forming a

sort of bulla on the surface. This becomes infected, breaks down, and forms an ulcer with a minute hole in the centre, through which part of the worm may protrude.

Complications and Sequelæ of Ulceration.—Cellulitis or erysipelas may attack the tissues about an ulcer. Hemorrhage from an ulcer is not uncommon, especially in varicose cases. It has been fatal. When an ulcer has healed, the resulting cicatrix may be the cause of serious trouble. Thus, it may lead to unsightly deformities and distortion when on the face or neck; when it is situated near a joint, more or less ankylosis may result. Keloid may also develop in the scar of an ulcer. Ulceration may lead to destruction of important structures, such as bones, cartilage, joints, muscles, and it may also cause infection of lymphatic vessels and nodes. Profound constitutional disturbance and weakness may result. Ulcers affecting the hollow viscera, such as the stomach and intestines, may perforate, giving rise, unless protective adhesions be formed, to fatal peritonitis. Chronic ulcers, or those which have healed, may cause stenosis of the lumen of the bowel.

Caries of Bone.—Analogous to ulceration of the soft tissues is the molecular disintegration of bone known as *caries.*

Caries is a chronic process of gradual softening or breaking down of bony substance, and is in all cases the result of inflammation. Infective agents are brought to the bone by the blood stream, and are deposited in the smaller vessels in the bone spaces. The ordinary phenomena of inflammation result, save that swelling cannot occur, owing to the unyielding nature of the tissue. Pressure, interference with the nutrition of the part, and the toxic emanations from the bacteria, all combine to bring about the death of the part. The products of inflammation are thrown off, mixed with calcareous matter and particles of decalcified bone in the form of sand or grit (molecular necrosis of bone—von Volkmann). Actual loss of substance thus occurs. Caries is met with most commonly and typically in connection with tuberculosis, syphilis, actinomycosis, acute and chronic osteomyelitis, and in suppurative processes extending to the bone. When there is a dry, cheesy sort of detritus produced without pus, we have what is known as *caries sicca.* Or, the carious process may so extend as to encircle a considerable area of bony substance, which it thus deprives of nutrition. As a result a large sequestrum is formed. This is termed *caries necrotica.* Molecular necrosis is met with more particularly in association with acute suppuration and where the granulation tissue is of low vitality, direct death of small particles of bone resulting. The finer details of the process in caries are largely a matter of speculation. Billroth thought that the essential factor was the resorption of the bone by the cells of the granulation tissue. Von Volkmann believed that the bone is disintegrated by the chemical solution of the ground substance with the liberation of the calcareous salts.

A similar obscurity befogs the subject of ulceration of the soft tissues.

Ulceration cannot take place in healthy tissues. There must be some previously existing disturbance which impairs vitality. If inflammation be not the disturbing factor in the first instance, it quickly becomes associated with the process. Breaking down of tissue is for a time at least in excess. How is the dead material disposed of? Two methods are conceivable. Either it may be disintegrated and cast off externally, or it may be absorbed. No doubt both methods are at work, but the former seems to be by far the more important. Inasmuch as material to be removed, such as portions of epidermis, fragments of bone or of soft tissue, are usually cast off rather than absorbed, we may infer with some certainty that the same general rule holds good in regard to ulceration and caries. It is, in fact, not uncommon to recognize small particles of bone or other tissue in the discharge from ulcers, while in certain cases considerable areas of dead tissue—*sloughs*, as they are called—are produced, which, when they are cast off, leave an ulcerating surface beneath. Thus we have all possible gradations between an impalpable disintegration of tissue (molecular necrosis, or ulceration in the strict sense) and the separation of visible particles or larger masses (sloughing or gangrene). The importance of the external discharge of dead material is seen particularly well in the case of abscesses, which may in a sense be regarded as concealed ulcers. Wherever possible, the pus burrows its way to the surface of the body or to some hollow viscus, and is there evacuated and removed. Healing in many cases will then occur spontaneously. If this do not occur, in many cases the abscesses are not absorbed, but go on extending. Indeed, only the smaller foci of suppuration can be removed by absorption, and that often imperfectly.

With regard to the question of absorption, we cannot altogether deny that it is of some importance. No doubt the excretions or discharges from certain ulcers, inasmuch as they contain enzymes derived from bacteria, are competent to bring about solution of the tissues, although it is not likely that this invariably occurs, as Rokitansky used to think. Now if such discharges be pent up, the disintegration process often proceeds apace, as we have so often opportunity to note clinically, and in some cases the products of disintegration, together with septic matter, are absorbed into the circulation, partly through the lymph stream and partly through the agency of the phagocytes. Proof of this is found in the cases of cellulitis, erysipelas, and septico-pyæmia which occasionally complicate ulceration. The removal of inflammatory products in other forms of inflammation—a process which is so constant an accompaniment in the process of repair—would induce us to think that a somewhat similar state of affairs is present in ulceration.

DISTURBANCES OF THE CIRCULATION.

The circulation of the blood throughout the body is carried on by means of a muscular force and suction pump—the heart,—with which is connected an elab-

orate system of more or less elastic tubes—the arteries, capillaries, and veins. Within the vascular system the blood pressure is dependent, first, on the force of the contractions of the heart muscle, and, secondly, on the amount of resistance manifested in the peripheral vessels. The blood pressure is greatest within the heart during systole, and falls gradually in the arteries, capillaries, and veins, in the order named. It is least at the venous orifices of the heart during diastole. The blood pressure is also governed to a large extent by the elasticity of the vessels and the degree of their tone. The amount of blood in any part depends, in addition to the influence of muscular action and elasticity, upon the vasomotor nerve mechanism, which determines the calibre of the vessels, their distensibility, and, hence, their capacity. The circulation is apt to be feeblest in those parts of the body which are most remote from the heart, and in the dependent portions. Under ordinary circumstances, the blood pressure and the amount of blood in any given part vary according to the nutritive and functional needs. The circulation may be deranged by causes which interfere with the onward flow of the blood and lymph. These may be systemic or local in their operation. Or the blood itself may be altered in amount or in quality.

Hyperæmia, or Congestion.—The amount of blood in any given part varies considerably even within physiological limits, according as to whether the function of the part is active or in abeyance. Should the amount exceed or fall below these limits, owing to causes other than physiological ones; or should the variation persist for an abnormal length of time, then we speak of pathological disorders of circulation. An excess of blood is called *hyperæmia* or *congestion;* lack of blood is called *anæmia*, or, more correctly, *ischæmia.*

Hyperæmia may be general or local. *General hyperæmia*, or *plethora*, as it is called, is rare, if it can be said to occur at all. Now and then we meet with individuals whose circulatory system seems to be overfilled. Especially do we see this in those who have died from obstructive heart affections. There seems to be more blood than usual in the body, although it is certainly not normal blood. During life, however, any excess in the total quantity of the blood is quickly compensated by increased activity of the emunctories.

Local hyperæmia may be due to an excessive supply of arterial blood—*active hyperæmia;* or to some obstruction to the outflow of venous blood—*passive* or *venous hyperæmia.*

Active hyperæmia results from a variety of causes, among which may be mentioned increased heart action, dilatation of the arteries of a part, stimulation of the vasodilator nerves, paralysis of the vasoconstrictor nerves, the diminution of extravascular pressure. It is seen particularly well in the first stages of inflammation. Irritations of all kinds, such as those produced by traumatism and by chemical, thermal, and mechanical influences, are competent to produce congestion. Local anæmia, when continued for any length of time, is usually followed by hyperæmia. The removal of long-continued pressure upon

blood-vessels is succeeded commonly by arterial dilatation and congestion. Thus, the application of an Esmarch bandage is followed by arterial dilatation on its removal. The sudden removal of fluid from the chest or abdomen is followed by local active hyperæmia, which may be so extreme as to cause faintness, owing to the collateral anæmia of the brain that results. Closure or narrowing of an artery leads to collateral hyperæmia of the adjacent parts. The pressure of tumors, of enlarged lymph nodes, or of inflammatory products upon the sympathetic nerve ganglia or fibres, sometimes causes arterial hyperæmia, owing to paralysis of the vasoconstrictor nerves.

A part affected by arterial hyperæmia, if on the surface of the body, is of a more or less deep red color, and is somewhat warmer than the surrounding structures. In many instances nutrition is stimulated, and probably function is increased.

Local venous hyperæmia, or passive congestion, is due to some interference with the outflow of the blood from an organ or tissue. The obstruction may be due to causes situated in the heart, mediastinum, or lungs, or to more strictly local and circumscribed conditions. A great variety of causes might be mentioned, such as the external compression of the efferent veins by tumors, aneurisms, ligatures, inflammatory infiltrations and exudates, cicatricial bands; closure of the lumen of veins by ingrowing tumors, thrombosis, or embolism; pressure in the abdominal cavity from tumors, effusions, and the pregnant uterus; constriction of the veins of the intestine by the neck of the sac in strangulated hernia.

The result depends, of course, upon the extent of the obstruction, its site, and the presence or absence of a collateral vascular system. If the part be supplied with anastomosing branches, occlusion of a vein is followed by only a temporary overfilling of the veins on the distal side of the obstruction. If, however, communication with other veins is slight or lacking, then more serious and lasting disturbance will arise.

The pathological changes which result from the complete obstruction of the return flow through the veins of an organ or tissue have been fairly well determined from experiment and clinical observation. The veins and capillaries on the distal side of the obstruction are greatly distended with blood. The distinction between the axial and peripheral currents in the veins is lost, the plasma gradually disappears, and the vessels become filled with closely packed red blood corpuscles. In an hour or two the blood has stopped in the veins and capillaries (stasis), and a few red cells find their way from the vessels into the neighboring tissue spaces. If there be any anastomoses with other vessels, capillaries hitherto unseen open up and an attempt is made at re-establishing the circulation. The effect of all this is that a rise of pressure occurs in the veins and capillaries, causing them to dilate. The blood becomes still more venous, owing to the stasis, and this interferes with the vitality of the endothelial cells

lining the vessels. Then, owing to the distention of the vessels and the lowered nutrition of their walls, transudation of the fluid part of the blood occurs. The process is in part compensated by contraction of the arterioles in the congested area, which is secondary to the diminished amount of blood flowing through the part. This tends to prevent an excessive increase in the intravenous pressure, to limit the amount of transudation, and to give time for a collateral circulation to be established.

To gross appearance, a region the seat of passive congestion is swollen, dusky red or purplish-red in color (cyanosis), cooler than normal, and its functional activity is diminished.

The final results of passive congestion depend upon the extent and duration of the condition. Temporary congestion may lead to no permanent changes. Congestion continued for a longer period causes pressure upon the cells, which become fattily degenerated, hydropic, and atrophied. In the more advanced conditions many cells, especially the more highly differentiated, such as the parenchymatous cells of glands, disappear and are replaced by fibrous tissue. The disintegration of the red corpuscles which have passed into the tissues leads also to the deposit of blood pigments (brown induration). In the most severe cases, where the circulation is absolutely and permanently stopped, hemorrhagic infarction of the part may occur, followed by gangrene.

Œdema.—Mention has been made of the fact that in passive congestion the plasma passes out from the vessels into the interstices of the tissues. Should the lymph circulation be inadequate to carry it away, it accumulates in the part and leads to the condition known as *œdema* or *dropsy*. This transudation and accumulation of the fluid portion of the blood may take place in various parts of the body. It may occur in the peritoneal cavity, and is then known as *ascites*. Effusion into the pericardial and pleural cavities is spoken of as *hydropericardium* and *hydrothorax*, respectively; into the tunica vaginalis, as *hydrocele;* into the subarachnoid space, as *external hydrocephalus;* into the ventricles of the brain, as *internal hydrocephalus.* Generalized œdema of the subcutaneous and intermuscular connective tissue is called *anasarca.*

Œdema occurs in the earlier stages of inflammation—hence called *inflammatory œdema*—and, as we have seen, in passive congestion. Three factors are of importance in determining the production of transudation into the tissues, namely, pathological variation in the blood pressure, alterations in the composition of the blood itself, and changes in the structure and function of the vessel walls. Whether œdema will result or not, in cases of transudation, depends entirely on the ability of the lymph channels to cope with the increased supply of fluid in the tissues. Obstruction to the current in the lymph vessels does not usually cause œdema, inasmuch as the anastomoses are very abundant, and any excess of fluid may be reabsorbed by the veins. Complete obstruction of all the lymph vessels of a part may, however, lead to a pure lymphatic œdema. The

same thing is seen in cases of obstruction of the thoracic duct (whether from tumors or from other causes), which results in what is known as *pseudo-chylous ascites*.

Increased pressure within the arteries does not give rise to œdema, provided that the return flow through the veins be unimpaired.

Increased pressure within the veins, such as occurs in passive hyperæmia, is, however, an important factor. Thus, œdema and effusions into the various cavities of the body are common in cases of general passive congestion, the result of obstructive valvular disease of the heart and of certain pulmonary and renal affections which interfere with the circulation. In such cases the œdema usually begins in the dependent or more peripheral parts, where we encounter the influence of gravity or of a weak circulation.

Local œdema may follow local passive congestion, as in the production of ascites in portal obstruction, or as the result of the pressure of tumors, inflammatory exudates, splints or other surgical appliances, on the veins of a part.

Increased pressure within the veins seems to predispose to transudation, owing to thinning of their walls and the presence of a *vis a tergo*. Probably, also, long-continued distention and imperfect nutrition lead to impaired elasticity of the extravascular tissues, so that the lymph tends to accumulate. More than this, however, seems to be necessary. Certain œdemas are met with in which the main condition appears to be some alteration in the secreting powers of the endothelial cells lining the vessels. Such are the œdemas formerly called hydræmic, and those due to the injection into the circulation of such substances as peptone and the enzymes of the various digestive secretions. At any rate, sufficient evidence has accumulated to show that transudation is not a mere question of pressures, filtration, and osmosis. The secretory activities of the cells lining the vessels must be taken into account as well. This leads us to conclude that there is not so much difference between the transudates and inflammatory exudates as used to be thought. The old view was, that transudates passed through the healthy vessel walls by a simple process of filtration or osmosis, while in the case of inflammatory exudates there were serious alterations in the vascular walls. This distinction cannot now be said to hold good, except in the most general way. The vital secretory powers of the vascular endothelium have to be taken into account in all cases.

Passive effusions or transudates are clear, usually colorless, of low specific gravity (from 1.006 to 1.012), and relatively poor in albumin. A few leucocytes and red corpuscles are usually present, and also swollen and fattily degenerated endothelium.

Inflammatory exudates are usually turbid, sometimes mixed with blood, of high specific gravity, and rich in proteids. Spontaneous clotting may take place. A relatively larger number of leucocytes is present. Inflammatory œdema is found in the neighborhood of inflammatory foci or may be caused

directly by the local action of various toxic, thermal, and traumatic agencies. It no doubt represents the first stage in the formation of inflammatory infiltration.

Œdematous tissues and organs present a characteristic waterlogged appearance, owing to the accumulation of fluid in the interstices. The part is swollen, pits on pressure, and, if an incision be made, clear, watery fluid exudes. On section, the tissue is juicy, of a semitranslucent appearance, and drips watery fluid. In the case of an extremity the skin is greatly stretched, is shiny, thin, and may present livid scars. In œdema due to passive congestion the part may be congested, at least in the earlier stages, but later becomes anæmic, owing to the pressure of the fluid.

Effusions into the body cavities lead to dilatation of the cavity, with compression of the viscera contained within, and, in time, to thickening of the serous lining.

Microscopically, œdematous tissues present some enlargement, with more or less dissociation of their elements. In the more extreme forms the cells and fibres are swollen, hydropic, and vacuolated.

The results of œdema depend upon its localization and extent. Effusions into the body cavities may lead to serious consequences, owing to pressure upon or dislocation of important organs. Transudation into the brain substance, or its ventricles, or into the subarachnoid space may lead to paralysis and death. Œdema of the glottis is a dangerous and often fatal complication of certain affections, such as Bright's disease, laryngitis, cervical cellulitis. Prolonged œdema of the skin and subcutaneous tissues leads to lowered vitality of the part, and may result in ulceration or gangrene. Infection readily occurs, and the condition may be complicated by erysipelas or cellulitis.

Anæmia.—The term "anæmia" literally means absence of blood. To a certain extent the term as ordinarily used is a misnomer, inasmuch as complete absence of blood does not occur in the animal organism, except in the most circumscribed areas. Further, we speak of a person as being "anæmic," when we mean that his skin and mucous membranes are pale and apparently bloodless. This pallor, however, need not depend on a complete or partial deficiency in the amount of blood in the part, but may be due to changes in the blood itself. The blood may be there in normal quantity, but may be lacking in red corpuscles or the corpuscles may be poor in hæmoglobin. It would, therefore, be more strictly correct to speak of a diminution in the total quantity of blood in the body as "ischæmia," and to keep the term "anæmia" for the purely local disturbances associated with deprivation of the blood supply to a part. Alterations in the quality of the blood should be given other designations. Probably, however, the word "anæmia" has been so long employed in this loose way that it will continue so to be used. It is well, however, to qualify it when necessary, so as to promote precision of language.

General systemic ischæmia will be discussed elsewhere, and we will confine our remarks here to the consideration of local anæmia and ischæmia.

The supply of blood going to a part may be diminished or cut off completely in a variety of ways. The total quantity of blood in the body may be less than normal, so that a smaller quantity reaches the various parts; or some local condition may prevent the blood reaching the part. Thus, the lumina of the afferent arteries, arterioles, and arterial capillaries may be diminished or occluded by pressure from without, spasm, or alterations in the structure of their walls. Local anæmia, for example, may be produced by the compression of an extremity by an Esmarch bandage, the ligation of the principal arteries, the pressure of a tumor, of an inflammatory exudate or effusion, or of a contracting, cicatricial band. The artery may also be more or less completely occluded by endarteritis, sclerosis, or the invasion of malignant growths; or its lumen may be obstructed by thrombi or emboli. Disturbances of the vasomotor system may produce local ischæmia. Brown-Séquard showed that stimulation of the cervical sympathetic is followed by contraction of the arterioles of the same side of the head. An excess of blood in any part, as in some cases of passive congestion, may result in a deficiency of blood in some other region. This is called *collateral anæmia.* A good example of this is seen in the ordinary "faint." Under the influence of pain, emotion, or fright, a nervous disturbance takes place, which determines large quantities of blood to the abdominal viscera. This leads to ischæmia of the brain and loss of consciousness.

In certain parts of the body, such as the heart, brain, spleen, kidneys, some portions of the long bones, and the retina, there are what are known as "terminal" or "end" arteries; that is to say, arteries which do not connect with anastomosing branches. Should such an end artery be occluded, complete anæmia of the part ordinarily supplied by that vessel will result. This leads to the so-called *anæmic necrosis* or *infarction.* The condition is met with typically in the kidney, where we find more or less wedge-shaped areas of an opaque, yellowish color, devoid of blood, and showing microscopically coagulation-necrosis. At the apex of the wedge can be demonstrated the occluded vessel. Such an infarct is called an *anæmic* or *white infarct.* Blood may, however, make its way in time from the neighboring capillaries into the anæmic part, thus converting the white into a *red* or *hemorrhagic infarct.* In the case of the brain, infarction is followed by colliquative necrosis (red or yellow softening). Infarcts are most commonly produced by emboli. If infection take place, we get suppuration in the affected part. If the affected part remain aseptic and if it be not of vital importance, the dead tissue is in time absorbed and replaced by fibrous tissue.

The results of a circumscribed anæmia depend upon its extent and upon the locality which it occupies. Complete deprivation of the blood supply in certain areas produces, as we have seen, death of the part. In other regions, where a collateral circulation is present or can be established, less disturbance is mani-

fested. Provided that the circulation be cut off for only a short time, no lasting results follow. More severe disturbance may result in minor degenerative changes and atrophy of the cells of the affected part. Where large vessels are obstructed it is not uncommon to find new channels of supply opened up, and previously existing anastomosing branches enlarge and dilate to meet the altered conditions of nutrition.

ALTERATIONS OF THE BLOOD.

These are chiefly of interest to the physician. They will, therefore, be dealt with here only in a sketchy way, but an attempt will be made to indicate, as far as may be, the bearing of disorders of the blood on surgical practice

In brief, the blood consists of a fluid part—the plasma—and certain formed elements—the red corpuscles, leucocytes, hæmatoblasts or blood platelets and "hæmokonien" or "dust bodies."

The blood may manifest abnormal changes in regard to its total quanti y. It may be excessive in amount (*plethora*) or diminished (*ischæmia, oligæmia*); or the relative proportions of plasma and corpuscles may be altered. Thus, there may be an absolute or relative increase in the plasma, while the corpuscles are normal in numbers and character (*hydræmia*); or, again, the plasma may be diminished in amount, so that the blood becomes more concentrated (*polycythæmia*).

Foreign substances may gain entrance into the blood, or substances which are normally present in small amount may be abnormally increased. Thus, bile, melanin, coal pigment, calcareous salts, fat, sugar, glycogen, toxic substances of many kinds, portions of tumors, necrotic tissue. gas, bacteria, and animal parasites may be found in the blood.

Finally, the corpuscles may be altered in number, both absolutely and relatively to the amount of plasma, or they may vary in their relative percentage to one another, or, again, in their quality and characteristics. The red cells may be diminished in numbers, as in most forms of anæmia, or increased. They may be deficient in hæmoglobin, as in chlorosis, or certain of them may contain an excess of this substance, as in pernicious anæmia. The leucocytes may be increased (*leucocytosis*), or diminished (*leucopenia*), in numbers; they may be altered in the proportions which one form bears to the other; or, finally, certain abnormal forms may make their appearance, as in leukæmia.

By an extension of the idea, the term "anæmia" is commonly employed to designate changes in the number and character of the corpuscles, as well as diminution in the total amount of blood. It is usual to divide the anæmias into *primary* and *secondary*. The primary are: chlorosis, pernicious anæmia, leukæmia, and pseudo-leukæmia. The secondary anæmias result from a great variety of causes, such as loss of blood, impaired nutrition, cachexia, the presence of intestinal parasites, infection, toxæmia.

The Primary Anæmias.—*Chlorosis.*—The main changes in the blood are as follows: The specific gravity is reduced. The number of the red cells is normal or nearly so. In neglected cases they may, however, sink to 1,500,000 per cubic millimetre (Stengel). Not infrequently they are increased (7,100,000 in one of Cabot's cases). The average would be about 4,000,000 or slightly over it. The diagnostic point is the diminution of the hæmoglobin out of proportion to the diminution of the red cells. It may be reduced to twenty per cent or under, but on the average is about forty-one per cent. The number of the leucocytes is about normal. Rarely, they are somewhat increased, especially during rapid convalescence. The hæmatoblasts are always increased. In severe cases the red cells are somewhat diminished in size. In mild cases the size is unaltered. Only in the severer forms is poikilocytosis observed. Rarely, normoblasts may be seen.

The disease is found almost exclusively in girls and young women, developing with the onset of puberty or shortly after. Thrombosis of the cerebral sinuses and of the veins of the extremities is a not infrequent complication.

Pernicious Anæmia.—The striking peculiarity in this disease is extreme diminution in the number of the red cells, with a relative increase in the amount of the hæmoglobin. The red cells usually fall to between 2,000,000 and 1,000,000 per cubic millimetre. The lowest count on record is in a case of Quincke's—143,000. The total amount of hæmoglobin is, of course, much below the normal, but is relatively increased per corpuscle. The color index may reach as high as 1.7. With regard to the size of the red cells, the average diameter is increased, but normocytes, microcytes, and macrocytes are to be seen. Usually poikilocytosis is marked. The formation of rouleaux is absent or slight. The blood clots slowly. Basophilic degeneration may sometimes be seen in the red corpuscles. Nucleated red cells, normoblasts, and megaloblasts are usually to be found in considerable numbers.

The leucocytes are usually greatly diminished in severe cases. There is a relative increase in the lymphocytes. Rare myelocytes may be met with. The blood platelets are diminished.

Leukæmia.—The most important of the primary anæmias, from the surgeon's point of view, is leukæmia. This disease is manifested in two well-marked forms, the *lymphatic* and the *myelocytic*. According to the predominating type—for mixed forms are not uncommon—the most striking external features are enlargement of the lymph nodes, enlargement of the spleen, and pain in the bones. For the relief of these conditions the surgeon is occasionally consulted, and, if unwary, may be led seriously astray, to the great detriment of the patient. A careful blood examination is called for in all cases of anæmia associated with enlargement of the lymph nodes or spleen, and will reveal the true nature of the case. Operative measures are uncalled for in such cases, and, in fact, are very liable to end fatally for the patient.

Leukæmia is characterized by the appearance, in the blood and tissues, of enormous numbers of leucocytes. According to the appearance of the blood and the condition of the organs, we can differentiate lymphatic leukæmia, both acute and chronic, and myelocytic. Mixed forms also occur. The cause is still unknown, but, whatever it may be, it certainly stimulates greatly the formation and cell division of the leucocytes, and increases the facilities for these leucocytes to enter the blood.

Acute lymphatic leukæmia, or acute lymphocythæmia, generally occurs in young persons between the ages of eleven and twenty-four. It may rarely be found in very young children, and has been observed at birth. It begins acutely with fever, a rapidly progressive anæmia, hemorrhages from the mucous membranes, purpuric spots upon the skin, and slight or moderate enlargement of the lymph nodes, spleen, and liver. The affection ends fatally in five or six weeks as a rule. Rarely, it may terminate in a few days. Occasionally it may last some weeks or even months, and then becomes chronic. Vomiting and diarrhœa occur, ulcerations take place in the mouth and gastro-intestinal tract, the patient becomes rapidly exhausted, and passes into a "typhoid" state, in which he dies. Delirium, convulsions, and coma may be observed.

Chronic lymphatic leukæmia, when typical, begins more gradually, with local manifestations in the form of enlargement of the various lymph nodes, to which general symptoms are superadded only in the later stages. The disease lasts some months or for several years; on the average, from nine months to two years. The nodes most often affected are the cervical, then the inguinal, retroperitoneal, mesenteric, and axillary. Ultimately all become involved. The spleen and liver are moderately enlarged. Moderate anæmia, emaciation, and progressive loss of strength are the leading features. Later, a tendency to hemorrhages into the skin and viscera, and from the various mucous surfaces, manifests itself.

With regard to the blood, there is a marked leucocytosis, the white cells reaching from 100,000 to 300,000 per cubic millimetre in the chronic form, and 100,000 or less in the acute. The leucocytosis is therefore much less than in the myelocytic type. The leucocytosis is further characterized by the enormous preponderance of the mononuclear forms. In healthy blood the lymphocytes are somewhat less than thirty per cent of the total number of leucocytes, but in lymphatic leukæmia they may amount to more than ninety per cent. In the acute form the large lymphocytes tend to predominate, while in the chronic it is the small.

Myelocytic Leukæmia.—This is the form of leukæmia most commonly met with, and is the usual type found in the adult. In the vast majority of cases it comes on gradually. The general health begins to fail, the skin becomes pale and muddy, there is a gradually increasing enlargement of the abdomen, and possibly a dragging pain in the left flank. A diurnal rise in

temperature may be the first symptom in some cases. Priapism may also be an early sign.

Myelocytic leukæmia is, as a rule, a chronic affection. Rarely it begins acutely with fever and hemorrhages. The result is invariably death.

The symptoms are at first slight, and patients usually seek medical aid some time after the disease has become well established. A sense of weight or actual pain in the abdomen, due to the enlarged spleen, is sometimes complained of, and the patient may himself discover the existence of a tumor in the abdomen. The progressive enlargement of the spleen is the most constant and conspicuous feature of the disease, and is often associated with enlargement of the liver. In such cases the protuberant abdomen contrasts greatly with the emaciation of the thorax. The various lymph nodes are usually slightly enlarged, but not obtrusively so. Tenderness over the bones is experienced in some cases. The skin and mucous membrane are somewhat pale and earthy in appearance, but the outward evidences of anæmia need not be striking. There are general lassitude and weakness, and there may be dyspnœa, palpitation of the heart, and faintness on exertion. The patient gradually emaciates, and there are occasional elevations of body-temperature. The average duration of the disease is from one to three years.

A rare form of myelocytic leukæmia, but one which undoubtedly occurs, is the so-called *myelogenous*, in which the spleen, lymph nodes, and liver are not enlarged, at least to physical examination, the characteristic changes of the disease being confined to the bone-marrow. The blood changes are, however, identical with those of the ordinary, or spleno-myelogenous, form.

The blood in myelocytic leukæmia shows a diminution in the red cells and a great increase in the white. The hæmoglobin content of the blood is diminished. The red cells usually average about 3,000,000 per cubic millimetre, but may be reduced to 1,000,000 or less. Ordinarily the white cells exceed in number 100,000 per cubic millimetre, and may reach 1,000,000 or even more. Cases have been known where the white cells were as numerous as the red.

The character of the white cells present in the blood is the most important diagnostic feature of this disease. Not only are the ordinary leucocytes increased in numbers, but certain abnormal forms, chiefly derived from the bone-marrow, make their appearance in great abundance. Three types of myelocytes, one or other of which may predominate, are to be found—the eosinophilic marrow cell, the neutrophilic marrow cell (Ehrlich's Markzelle), and the marrow cell of Cornil. Occasionally cells with coarse basophilic granulations may be found (Mastzellen). All the forms just mentioned are mononuclear. Dwarfed forms of the various white cells are often to be found. The red cells usually show all the changes peculiar to a severe primary anæmia. Nucleated forms, generally normoblasts, but also megaloblasts, may be noted. Basophilic degeneration also occurs, and sometimes there is poikilocytosis.

It is important to remember that an intercurrent inflammatory process may greatly modify the blood picture of leukæmia. In such cases the tendency is for the blood to approximate to the type of the ordinary febrile leucocytosis. The total number of leucocytes is diminished, and the ordinary polymorphonuclears begin to predominate. Nucleated red cells and myelocytes are, however, never entirely absent. Under the continued use of arsenic, too, there may be at times a similar reduction in the number of the white cells, and the blood picture may change to one closely resembling primary pernicious anæmia. Such remissions, if such they may be called, are but temporary.

The cause of leukæmia is as yet unknown. Opinions are also divided as to whether the disease is allied to the malignant tumors or is to be regarded as a specific infectious leucocytosis. The enlargement of the spleen is not an essential feature of the disorder, nor is this organ primarily at fault. Therefore removal of the spleen, as has been advocated and practised, is unscientific and unjustifiable. The operation is, moreover, usually fatal.

Pseudo-leukæmia, or Hodgkin's disease, occasionally comes under the observation of the surgeon, with a view to possible operative interference. To external appearance this disease is practically identical with chronic lymphatic leukæmia, but the characteristic blood changes of the latter affection are not present. In Hodgkin's disease there may be a polymorphonuclear leucocytosis or even a slight lymphocytosis. Extreme lymphocytosis is not observed. Inasmuch as patients suffering from Hodgkin's disease occasionally develop leukæmia, there is some reason for thinking that leukæmia and pseudo-leukæmia are simply phases of one and the same pathological process. The surgeon may be called upon, in Hodgkin's disease, to remove nodes which are pressing upon important structures.

The Secondary Anæmias.—Besides the grave forms of anæmia just referred to there are others, less severe and often temporary, which result from affections other than those of the blood itself or blood-forming organs. The causes are very varied. Chief among them are hemorrhage, malignant disease, chronic suppuration, acute and chronic infectious diseases, nephritis, dysentery, heart disease, toxæmia of all kinds, prolonged lactation, myxœdema, rickets, Addison's disease, and any condition which leads to disintegration of the blood cells.

In general, it may be said that the red corpuscles are more or less diminished in numbers. The red cells may be deformed or of small size. In some severe cases the blood may resemble in many particulars that of chlorosis or that of pernicious anæmia. Normoblasts occur, but are scanty. Megaloblasts are encountered still more rarely. Karyokinesis and karyolysis may be observed.

Leucocytosis may or may not be present. The white cells are usually increased in number in cases of malignant disease, in tuberculosis with ulceration, and in suppurative processes generally. The increase is, as a rule, in the polymorphonuclear form.

The Anæmia from Hemorrhage.—The subjective and objective phenomena of sudden and extreme loss of blood are well known and need not be described here. With regard to the blood itself, there is, of course, a more or less pronounced diminution in its total quantity, involving all its components. The red cells are diminished in numbers, and there is a corresponding reduction in the amount of hæmoglobin. If the hemorrhage be recovered from, the loss in bulk of the blood is compensated by a reabsorption of plasma from the interstices of the tissues and by contraction of the vessels. The blood thus becomes more watery. The red cells are not altered in appearance. Shortly after the loss of blood, a tendency toward restoration of the former condition manifests itself. The red cells gradually increase in numbers, and young forms, microcytes and macrocytes, make their appearance, with also nucleated forms. The hæmoglobin is not so quickly replaced as the cells, so that many of them appear to be chlorotic. Very shortly, too, after the occurrence of the hemorrhage, regenerative changes become strongly marked in the leucocytes, which are notably increased in number. This post-hemorrhagic leucocytosis is a constant and characteristic feature. The blood thus gradually becomes more concentrated, and finally assumes its normal condition.

The length of time required for complete return to the normal depends upon the amount of blood lost, the age and idiosyncrasy of the patient, the character of his food, medicinal measures, and so on. Small losses of blood may be repaired in from two to five days; larger ones may require a month. Young children stand hemorrhage badly and take longer to recover.

The blood picture after repeated small hemorrhages is quite different. Such hemorrhages may be the result of nose-bleeding, hæmoptysis, hæmatemesis, melæna, hemorrhoids, hæmophilia, the hemorrhagic diathesis, certain uterine disorders, intestinal parasites. Small hemorrhages, as we have seen, are quickly compensated and repaired. Should they be repeated before restoration to the normal can take place, we get the picture of chronic anæmia. The extent of this will, of course, depend upon the number of the hemorrhages, their severity, and the vitality of the patient. The main changes may be summed up as follows: The red cells are diminished in numbers and the blood becomes more watery; the hæmoglobin is diminished in proportion to the diminution of the red corpuscles, but may be even more reduced in severe cases; microcytes, macrocytes, and poikilocytes may be found; nucleated red corpuscles are not uncommon; the red cells show sometimes polychromatophilism; the leucocytes (usually the polynuclear) at first are often increased, but in advanced cases may be diminished. In the most extreme cases the blood may resemble very closely that of primary pernicious anæmia.

Leucocytosis.—Leucocytosis is an increase in the number of leucocytes present in a given quantity of blood removed from a peripheral vessel, above the number normal for the individual concerned. Leucocytosis usually concerns

the polymorphonuclear leucocytes, but the others, lymphocytes and eosinophiles, may be at times affected. Therefore, in determining leucocytosis it is wise to make a differential count of the forms present.

Leucocytosis may be physiological or pathological.

Physiological leucocytosis is met with in the young infant, and also in adults during digestion, during pregnancy, just before death, after violent exercise, after massage, after short cold baths or prolonged hot ones.

Digestion leucocytosis amounts to an increase of about one-third in the number of the white cells. The normal percentages of the various cells may remain the same, or the lymphocytes may be absolutely or relatively increased. In chronic gastric troubles and gastric cancer, digestion leucocytosis may fail to occur.

In infancy the lymphocytes are relatively and absolutely increased—a fact which should not be forgotten in the examination of those of tender age.

Pathological leucocytosis may be due to hemorrhage, inflammation, intoxications, infection, or malignant disease, or to the exhibition of certain chemical substances.

Post-hemorrhagic leucocytosis has already been sufficiently dealt with.

Inflammatory leucocytosis is met with in inflammations of almost all kinds. Its degree seems to depend upon the balance existing between the inflammatory process and the resisting power of the patient. The character of the infection is also important. Leucocytosis is most marked in lobar pneumonia; moderate in most infective processes, including suppuration due to pyogenic cocci; but may be absent, as in typhoid fever, malaria, influenza, measles, acute miliary tuberculosis, leprosy. The occurrence of leucocytosis in a disease which ordinarily should present none may at times be of value to the surgeon, in indicating some suppurative complication. The increase of the leucocytes in inflammatory affections is usually in the polynuclears.

Extensive malignant disease is associated with an increase in the number of the polymorphonuclear leucocytes. This may possibly be due to ulceration and secondary infection, at least in many cases.

Toxic leucocytosis is met with in poisoning by ptomaïns, illuminating gas, ether, quinine.

The administration of drugs, such as the salicylates, pilocarpine, ergotin, the antipyretics, and tuberculin, and the injection of normal saline, will raise the number of the white corpuscles.

Lymphocytosis may be found at times in rickets, syphilis, scurvy, malaria, chlorosis, pernicious anæmia, malignant disease, and cachexias.

Eosinophilia, or increase in the number of the eosinophiles, is found in some diseases due to animal parasites, such as trichinosis and ankylostomiasis, and in malaria; in osteomalacia; in some cases of sarcoma; in certain skin diseases, as pemphigus, pellagra, dermatitis herpetiformis.

ON CERTAIN PRODUCTS OF INFLAMMATION.

Under this heading we propose to deal with the question of the origin and nature of inflammatory exudates, more especially in regard to lymph and pus.

The proper apprehension of this subject presupposes a knowledge of the principles exemplified in the process of inflammation. As this phase of the matter has been thoroughly discussed in article No. 1 we may proceed at once to the consideration of the question in hand.

Perhaps the simplest form of inflammatory exudation is that known as *serous exudation*. Instances of this are met with in the fluid contained in blisters produced by cantharides or by heat, in the effusion into joints or hernial sacs, which have been injured and become immediately inflamed. Such effusions are rare in inflammation, except in its mildest form, but are a common result of passive congestion. As we have already seen, there is no essential difference in composition between the so-called passive effusions or transudates and inflammatory exudates. The only practical distinction, and that a somewhat rough one, is that the latter contain more cellular elements and are much richer in fibrin-forming substances. The fluid from a simple transudate will not usually clot on removal from the body, while an inflammatory exudate will. At most, in passive effusions of long standing we may find a few flakes of fibrin floating, and even here it is open to assume that a low grade of inflammation has been present. We have to recognize, therefore, that there are many intervening stages between a simple serous effusion, the result of a passive transudation, and the cellular, more fibrin-containing exudate characteristic of inflammation. In many of these serous effusions, so long as the fluid remains within the body, clotting does not occur; but, as soon as it is withdrawn, fibrin is formed in considerable amount. Again, we have inflammatory exudates, and these are the commoner and more characteristic ones, which are so rich in fibrin-forming substances that when removed from the body they clot promptly and firmly, or they may even clot within the body. These exudates constitute what is usually known as *plastic* or *coagulable lymph*. The term "lymph" is somewhat unfortunate in this connection, as it is apt to lead to confusion with the lymph which flows within the lymphatic vessels. This, of course, has nothing necessarily to do with inflammation. By prefixing the word "inflammatory" we probably render the meaning sufficiently evident, and long usage has by this time sanctioned the error.

Plastic or Coagulable Lymph.—This is the exudation produced in what may be called "healthy" or "constructive" inflammation. It is in the main a fluid having properties not unlike the liquor sanguinis, more particularly in that it tends to coagulate. The amount of fibrin-forming substances is, however, somewhat variable, being at one time, as we have seen in the so-called serous exudates, comparatively trifling; at another, sufficient to produce a dense, thready, firm, coagulum, even during life. The number of the contained corpuscular ele-

ments, or leucocytes, is also variable. These are practically absent, or at least quite scanty, in serous exudations, more abundant in the ordinary inflammatory exudations, so that in some cases it is difficult to distinguish the exudate from pus, save in its faculty for coagulating. Such lymph is met with on the surface of recent wounds, in the neighborhood of many inflammatory foci, on abraded surfaces, and upon mucous and serous membranes. In the case of the serous cavities, where considerable quantities of exudation may collect, we can differentiate several forms, according to the relative proportions of the fluid part and the cellulo-fibrinous material. Thus, if the exudate be largely fluid, we speak of a *serous* exudate; if mixed with considerable fibrin and more numerous cells, we speak of a *sero-fibrinous* exudate; if mainly fibrin and cells, we have a *plastic* or *fibrinous* exudate.

Inflammatory exudates are not without import, and, indeed, subserve many useful purposes. First, the passage out of fluid and cells through the vessel walls lessens the congestion and diminishes the distention of the vessels of the inflamed part; secondly, the exudate tends to dilute any irritating substances, such as bacterial toxins or disintegrating tissue, which may be present; thirdly, it tends to flush out the part, and, by reducing toxic materials to a soluble and labile form, to promote the removal of such irritating substances from the part through the lymphatics and blood-vessels; fourthly, it in some cases helps to limit the inflammatory process; fifthly, in the case of uninfected wounds it forms a bland and unirritating natural dressing and tends to promote adhesion of inflamed surfaces and hasten the reparative processes; lastly, in certain cases it appears to possess bactericidal properties.

The importance of coagulable lymph in connection with the repair of injuries can be well seen in the study of a simple incised and uninfected wound, such as may be inflicted by the surgeon's knife. The first effect of the incision is to divide the tissues, vessels, nerves, and other structures of the part. At first a little blood will be effused from the severed vessels. This soon stops unless a large vessel be cut and left unsecured. Now when the cut surfaces are brought into close and accurate apposition, we have an instance of a reparative inflammation with the least possible amount of reaction. The divided cells for the most part die, at least if their nuclei be destroyed; the injured vessels retract, close, and become thrombosed, and an outpouring of serum takes place, which glues the two surfaces together and exudes slightly upon the surface as a clear, transparent fluid, that ultimately dries into a delicate membrane of fibrin known as the "scab." This effused serum is an admirable medium for the growth and development of new cells, as it contains all the necessary pabulum. Leucocytes pass out from the vessels into the injured region and into the lymph, so that in a few hours there may be a considerable aggregation of granular cells. Next, by a process not as yet fully understood, the adjacent capillaries send out buds from their sides, which gradually become hollowed out and permeable for blood. These

extend into the effused lymph until they meet similar buds from other vessels, with which they unite, forming loops of vessels. With them certain cells also invade the part, derived to some extent from the proliferation of the pre-existing connective-tissue elements about the walls of the vessels, or possibly, as some hold, also from the germination of the leucocytes. These are known as fibroblasts, and in time they become converted into dense, fibrous connective tissue. Thus, the jelly-like cement substance originally present is transformed by the process of *organization* into a firm connecting substance, *cicatricial tissue*, which binds the formerly dissevered surfaces together and to this extent makes good the injury. This process is called union by "*first intention*." As a rule, the more highly specialized cells, such as nerve cells or those of glands, are only incompletely restored, if at all, and the damage is repaired by more or less inert connective tissue. This ultimately contracts, many of its vessels become obliterated, and we get a dense, white *scar* or *cicatrix*. Where the epidermis is severed, the epithelial cells grow inward over the wound, and its continuity is restored in all parts.

In the case of an abrasion or superficial wound, when of limited extent, the exuded fluid coagulates upon the denuded surface, where it dries, forming a tough, somewhat flexible crust or scab. The effused lymph is gradually organized in the way just described, and the epidermis grows inward beneath the scab, which eventually falls off, leaving a slightly reddened, smooth surface, that in course of time pales and becomes scarcely distinguishable. This is known as *healing under a scab*, and is a very simple and effective method where it is practicable. If micro-organisms be present, as they so often are, healing by the two methods described may still go on, provided that the infecting organisms are of low virulence and of the non-pyogenic variety. Very frequently, however, as we shall shortly see, bacteria greatly interfere with the process of healing.

A more severe type of inflammation is that met with in connection with serous membranes, such as the peritoneum, pleura, and pericardium. Generally bacteria are at work in these cases. If we take, for example, a simple peritonitis, what happens is this: Bacteria reach the membrane attacked through the blood or lymphatic vessels. We get all the phenomena of inflammation—congestion, stasis, the passage out of plasma and corpuscles. The lining endothelium of the membrane becomes swollen, and the cells composing it desquamate and undergo hydropic and fatty changes. Next, the exudate appears upon the surface, usually at the points where the adjacent coils of intestine come into contact. At such places the membrane is reddened, swollen, and covered with yellowish-white fibrin and a variable amount of fluid. If much fluid be present, it collects in small pockets between the coils of intestine, in the flanks, and behind the liver, or may lie free in the pelvis and general abdominal cavity. In the course even of a few hours the inflamed surfaces become somewhat sticky and tend to adhere. Should the patient recover, organization of the lymph takes place as above

described, with the development of new blood-vessels and the formation of fibrous bands between the coils of intestines or between the abdominal walls, diaphragm, and underlying structures. Such adhesions are surgically of importance, since in some cases they lead to pressure upon important structures, obstruction of ducts, dislocation of the viscera, strangulation and incarceration of the bowel, and so on. In some cases where the inflammation has been prolonged, we may get firm, dense, whitish, almost cartilaginous plaques, surrounded by more fibrous adhesions which seriously hamper the working of the various organs.

In superficial inflammation of mucous membranes we get similar exudates, but mixed with mucin and often with blood. The reparative powers of the epithelium on mucous surfaces, especially that of the intestines, are not inconsiderable, so that the restoration of the normal structure of the part is often complete or nearly so. We see, moreover, in the case of mucous membranes especially, the exudation of a fluid which clots both upon the surface and in the interstices of the inflamed part. With this, apparently under the influence of bacterial toxins, or the interference with the circulation, or both, we get necrosis of the superficial part, with the formation of what is known as a "croupous" deposit or *pseudo-membrane.* This is met with notably in diphtheria.

We have hitherto been dealing with what may be termed "reparative" or "constructive" inflammation, the process by which a great variety of simple injuries are healed, and which illustrates also in typical fashion the *vis medicatrix naturæ* so often referred to by the older writers. The result of inflammation may not, however, always be so happy. Owing to the nature of the injury, the influence of external deleterious factors, or impaired vitality of the affected part, we may get a much more serious train of symptoms, characterized in the main by the *destruction* of tissue. Here we have to assume some cause at work which interferes with or interrupts normal local nutritive reaction. The most striking instance of this is *suppurative inflammation,* or *pus formation.* In the case of healing by first intention, as we have seen, there is practically no loss of tissue substance, or at least it is comparatively trifling. In suppuration, while in the end it may resolve and heal, as in the process of granulation known as healing by "*second intention,*" there is invariably a destruction of substance commensurate with the intensity and duration of the suppurative process.

Instances of suppurative inflammation might be multiplied almost beyond number, since this condition is one of the commonest with which the surgeon has to deal. As we have seen, the smaller abrasions and superficial injuries of certain tissues will, under favorable circumstances, heal with a minimum of reaction, as do aseptic incised wounds. Abrasions of the skin or mucous surfaces—a condition which implies a loss of the superficial epithelium—are from their situation particularly liable to infection, so that an inflammatory lesion is not infrequently converted into a suppurative one, if, indeed, it does not have that character from the beginning. We see this, for example, in abrasions caused by heat,

moisture, friction, desiccation, and cracking, in the rupture of vesicles and pustules, and in the excoriations from irritating discharges. In many cases superficial inflammation is transformed into one of a penetrating and destructive nature (ulceration). Suppuration may, again, take place in the deeper tissues and in parenchymatous organs. Here it may be diffuse or localized.

Perhaps the simplest form of suppurative inflammation is to be found in what is called healing by "granulation" or "second intention."

If, for example, we take the case of an open wound, such as might be caused by the operative removal of tissue, trauma, gangrene, or similar cause, we find that the process of healing proceeds in an orderly way, much as it does in the case of healing by first intention, except in so far as it is modified by the physical conditions existing. If the loss of substance be so great that the denuded surfaces cannot be coapted, or if there be some foreign material present which prevents proper closure, healing by simple adhesion is impossible and repair has to be brought about in a more indirect and tedious fashion.

The first manifestation of reaction, after bleeding has ceased, is the covering of the raw surfaces with plastic lymph. This acts as a protective varnish, lessens irritation, and forms a suitable pabulum for the growth of cells. The next step is that, with a certain amount of pain and swelling, and more or less systemic disturbance, the jelly-like lymph is transformed into a reddish, highly vascular, velvety substance—*granulation tissue*—which is bathed in a bland, yellowish, creamy fluid—*pus*. The granulation tissue is the same as granulation tissue everywhere else. It is composed of aggregations of cells of embryonic type, which have the power of developing into fully formed connective tissue, enclosing a network of capillary loops. Healthy granulation tissue is smooth and firm to the touch, of jelly-like consistence, and of a more or less reddish color, according to the amount of blood it contains. It bleeds upon the slightest touch, owing to the great number of delicate vessels which it contains. It is, moreover, covered on the surface with small elevations of varying size, containing minute vessels—*granulations*,—the spaces between which are filled with pus. The appearance of these granulations is a valuable index to the nature of the inflammatory reaction. When the reparative power is weak, the granulations are large, pale, and translucent, while the pus is thin and watery. When the part has been irritated, as from friction or unsuitable applications, the granulations are small and excessively red. Sometimes, where these causes persist, the granulations may disappear in places, leaving grayish points where they have died, or smooth patches where they have ceased to grow. Under certain abnormal conditions, not well understood, granulations become exquisitely painful, although ordinarily they are not sensitive. Again, wherever from any cause cicatrization is being obstructed, the granulations increase in size and may coalesce into a fungous mass, popularly known as "proud flesh." We find this sometimes occurring about a seton, a group of ligatures, a drainage tube, or in a wound involving the

sheath of a tendon. Such redundant granulations are more pale and flabby than are healthy ones. Under all circumstances the main feature of granulation tissue is its capacity for assuming a higher condition, namely, that of cicatricial tissue. Whenever healing is prevented, the complete transformation does not take place, and the granulation tissue will remain indefinitely *in statu quo*, ready, however, at any time to fulfil its destiny when circumstances render this possible.

The purulent exudation in these cases may be regarded as an excretion from the vessels,—an exudation peculiarly rich in leucocytes, and containing an admixture of fibrin and embryonic cells. Its presence is the cause of the heat and tension which are early manifestations in the injured region, as well as of the fever and malaise, should such there be; for, as soon as the exudation begins to appear externally, these symptoms begin to lessen.

It is a peculiarity of granulating surfaces to adhere if brought together and held in quiet contact, and this property is often taken advantage of by the surgeon to promote healing. This method is known as healing by "*secondary adhesion.*"

In the ordinary course of events, if the opposing surfaces cannot be entirely brought into contact, the granulations go on increasing in number, until they finally coalesce at the bottom of the wound, which is then gradually filled up. Coincidently with this, the granulations are converted into connective tissue, the superficial epithelium proliferates and closes in over the surface, and the loss of substance is repaired as far as may be by the formation of a dense, white cicatrix, which ultimately contracts and leads to more or less distortion of the part.

Occasionally, this sequence of events is interfered with and the result is less satisfactory. If the loss of substance be excessive, it may be beyond the power of the organism to repair it, and we have a granulating surface, persisting indefinitely without any effective attempt at cicatrization. A state of affairs somewhat like this is not infrequently met with in the case of superficial burns of great extent, which do not close in until skin-grafting has been performed. Or, a granulating wound may become infected, as in hospital gangrene and phagedæna, the granulations disappear, and the process is converted into one of ulceration. In some cases the base of the wound dies and is converted into what is known as a *slough* or *moist eschar.*

The Nature and Origin of Pus.—Healthy pus—*pus bonum vel laudabile* of the older writers—is a creamlike fluid, of a yellowish-white color, sometimes having a slightly greenish tint, with a faintly animal odor, a salty and sweetish taste, and a soapy, unctuous feel. When allowed to stand for some hours, pus separates into two parts—a thick deposit composed of leucocytes (*pus corpuscles*), embryonic cells, tissue débris, fat globules, and sometimes fatty acids, cholesterin, or blood; and a supernatant serous portion, the *liquor puris.* The liquor puris is a clear, slightly alkaline, albuminous fluid, without solid particles.

Microscopically, the solid constituents of pus are found to consist, in the

main, of leucocytes, adult and immature, some of which, in fresh pus at least, are healthy and manifest amœboid movement, while others are fattily degenerated or are dead.

In all forms of inflammation, except possibly the very slightest, constant features are the migration of leucocytes to the injured part, under the influence of the obscure force commonly designated positive chemotaxis, and the exudation of fluid and cells from the vessels (diapedesis). These phenomena are in a measure a response to the increased nutritional demands of the damaged tissues. The exudate, however, is very commonly produced in excess of the needs of the part, and therefore, if in the deeper tissues, will tend to accumulate there; or if nearer the surface, as in a granulating wound, will make its way through the layer of granulation tissue to the surface and be discharged as purulent exudation. The formation of healthy pus is to some extent an indication of the efficiency of the reparative processes, and we can thus understand what the older surgeons meant by the term "laudable" pus, pus being an almost constant accompaniment of every wound and surgical operation. But, as Listerism has abundantly shown, pus formation is by no means a necessary accompaniment of the repair of injury.

Some debate has arisen with regard to the question whether the presence of bacteria is essential to the formation of pus. Councilman and others have been able to produce suppuration by the introduction, into the subcutaneous tissues of experimental animals, under strict aseptic precautions, of such substances as turpentine, croton oil, metallic mercury. Subsequent inoculation experiments showed that in many instances pus so produced was sterile. Some recent experiments of W. W. Ford's, indicating that healthy tissues and organs contain bacteria normally, may be held, if accepted, to invalidate this conclusion. For there may have been present, in pus produced by injection of turpentine, microbes which failed to grow later, owing to attenuation. However this may be, pus, as met with by the clinician, practically always contains microbes, either dead or alive. And, as is well known, many bacteria, when injected into the tissues under suitable conditions, are competent to produce a suppurative inflammation. Such micro-organisms are usually called *pyogenic.* Among them are the Staphylococcus pyogenes aureus, commonly found in acute abscesses, the Staphylococcus pyogenes albus, flavus, and citreus, the Streptococcus pyogenes, Bacillus pyocyaneus, the gonococcus of Neisser, and the meningococcus. Under certain circumstances other bacteria, not ordinarily pyogenic, may produce pus. Such are the B. coli, B. typhi, the pneumococcus.

The Varieties of Pus.—*Healthy* or *laudable* pus has just been described. *Sanious pus* is that which contains blood. *Curdy pus* contains particles of fibrin or cheesy matter floating in it. *Ichorous pus* is of a watery character. *Muco-pus* contains mucus. *Sero-pus* is rather less rich in corpuscles than is healthy pus. *Blue pus* is pus of a bluish or bluish-green color, which is occasionally met with.

This peculiarity is due to the presence of the B. pyocyaneus, a chromogenic bacillus of rather low virulence. *Red pus* is due, according to Ferchmin, to a special microbe, which is non-motile, has rounded ends, and produces a reddish pigment on nutrient media.

The Forms of Suppuration.—Suppuration may be superficial, involving cutaneous, serous, or mucous surfaces; or deep, affecting subcutaneous structures or parenchymatous organs. As examples of the former we may take the suppuration which attacks infected wounds, abrasions, or losses of substance, of which we have just considered, as the simplest type, healing by granulation or by second intention. In the case of the serous membranes, as in empyema, pyopericardium, and purulent peritonitis, the process is not dissimilar, except that "granulations" do not play so obvious a part. Under the influence of some irritant the lining endothelium swells, the cells become cloudy or fatty, and eventually are desquamated. Then effusion from the vessels of the membrane takes place, first into the interstices, and then upon the surface, where it is deposited as plastic lymph, rapidly becoming highly corpuscular. The amount of effusion is apt to be large, and it collects at the bottom of the serous sac, separating the two layers of the membrane and often compressing the contained structures. Resolution takes place in part by reabsorption of the exudate into the vessels, but in larger measure by removal through the lymphatic channels. The vessels regain their normal tone, the exudation disappears, and the endothelium is regenerated. Adhesion of the two layers of the serous sac is particularly apt to occur.

Suppuration in the deeper tissues may be *circumscribed* or *diffuse*. A good example of the former is the ordinary *abscess*. An acute abscess exemplifies in a typical way all the phenomena of a severe inflammation, terminating in suppuration and destruction of tissue. We have the classical symptoms of redness, swelling, heat, and pain, together with, in some instances, constitutional disturbance, as headache, fever, rapid pulse. The cause is some injury, tissue degeneration, or infection, which leads to irritation of the part. There result, thereupon, increased afflux of blood to the part, distention of the vessels, and exudation, first of lymph and then of corpuscles. Owing to the local disturbance, the increased fluid thus brought to the part cannot be successfully disposed of. It accumulates, causing pressure upon the neighboring cells, separation of muscular and connective-tissue fibres, rupture of fibres, vessels, and nerve filaments. The exudation gives rise to the swelling, and the pressure upon the nerve fibres produces the pain. Thus in time the pus forms a cavity for itself. If the process do not soon come to an end, the pus generally burrows through the tissues along the lines of least resistance, and makes its way to the surface of the body or into some hollow viscus. This event generally results in healing by ordinary granulation, and is often imitated by art. According to clinical observation, the *raison d'être* of suppuration would appear to be an attempt on the part of the

organism to get rid of offending material, whether dead tissue, foreign bodies, bacteria, or other irritating substances. The process in the so-called "*cold*" or *chronic abscess* is identical, except that it is more sluggish. Swelling is present, owing to the exudation, but the redness, heat, and pain may be trifling or absent.

Diffuse suppuration is met with in the so-called phlegmonous inflammations, erysipelas and cellulitis. Here, the condition is for a time at least progressive, involving widely connective tissue, fascia, and tendon sheaths. Local œdema is marked, but the inflammation does not tend to become circumscribed, as in the case of an abscess, while the systemic manifestations are much more severe. General septicopyæmia not infrequently results.

The Results of Suppuration.—Suppurative inflammation may heal if it be not extensive, leaving little or no traces, or, when more severe, it will result in the destruction of the finer structure of the part, the elements of which are replaced by connective tissue that has no other function than that of restoring the continuity of the part and binding it together. Healing will more readily take place if the inflammation be well localized and so situated that the external discharge of the pus, whether naturally or by intention, is possible. Collections of pus in the deeper tissues may be absorbed and the destroyed part heal up with the formation of a cicatrix, or the pus may become inspissated or infiltrated with lime salts. In some cases, especially those of long standing, where the pus is retained, it leads to condensation of the neighboring tissues, with the formation of a sort of fibrous capsule, lined on the inner surface with granulation tissue—*pyogenic membrane.* This membrane is the result rather than the cause of suppuration, as used to be held. When the pus is deeply seated and burrows widely, it attempts to make its way to the surface and produces long tracks of communication with other parts, known as *sinuses.* When the pus is discharged upon the surface or into a hollow viscus, we get a channel of communication known as a *fistula.* This is, strictly speaking, a tube which is open at both ends; but the term is ordinarily used to designate any passage connecting the suppurating focus with the external air. Suppuration within joints or in serous sacs frequently leads to adhesion of the adjacent structures, with partial or complete occlusion of the cavity, compression of important organs, fibrous ankylosis, and the like. Calcareous deposits are not infrequent, and even new bone may at times be formed. Prolonged suppuration, as a remote sequel, not uncommonly results in amyloid transformation in the various viscera. A most serious result is the entrance of pus with its contained micro-organisms into the blood, with a general infection of the system.

PROCESSES OF REPAIR.

By EDWARD H. NICHOLS, M.D., Boston, Mass.

After an injury which causes a loss of substance of any of the tissues of the body, a series of changes occur about the injured area which make good the loss. In most tissues the loss is replaced to a very limited extent by newly formed tissue which completely restores the integrity of the original tissue. In such cases the process is spoken of as a process of *regeneration.* In most cases the destroyed tissue is replaced by tissue composed of newly formed blood-vessels and dense connective tissue, *i.e.,* by scar. This process is spoken of as a process of *repair.*

I. REGENERATION.

Regeneration means the replacement of lost tissue by newly formed tissue which has the same structure and function as the original. The power of regeneration varies enormously with different tissues and with the age of a given tissue. The younger a tissue is, *i.e.,* the more completely the tissue approaches embryonic tissue, the greater is its power of regeneration. If, for instance, the tail of a tadpole be cut off, it will be completely regenerated, muscle, cartilage, and epithelium appearing in their regular places. If the limb of a frog, however, be cut off, the limb will not be regenerated.

The less highly organized an organism, the greater its power of regeneration; *e.g.,* the unicellular organisms may be cut into several parts and a complete organism may be produced from each portion.

The more highly complicated a structure is, the less is its power of regeneration. Thus, when large areas of skin are destroyed the epithelial covering may be completely replaced by regeneration of epithelium; but the hair follicles and sebaceous glands, composed of epithelial cells with complicated arrangement and more highly differentiated function, are not replaced. In the same way, if single cells, *e.g.,* in the tubules of the kidney, are destroyed, they are replaced by a new formation of cells from adjacent cells; but the destruction of any considerable area of kidney substance is not completely restored by regeneration.

The more highly differentiated a tissue is for special functions, the less is the power of regeneration of that tissue; *e.g.,* in complicated glands there is greater power of regeneration in the ducts of the glands than there is in the specialized cells of the secreting portion. In the cells of the highly specialized central nervous system the power of regeneration is extremely limited. It also is doubtful whether there is any power of regeneration in striated muscle cells.

When tissue is regenerated to replace a loss, the law is that the newly formed tissue is produced from similar tissue; *e.g.*, epithelium cannot be reproduced from connective tissue. The cells of the new tissue arise by division of the old cells of similar tissue. This division of old cells to form new ones is a complicated process, and is spoken of as "mitosis," "karyokinesis," or "indirect cell division." (See Fig. 74.) The living cell consists of a nucleus, surrounded by a nuclear membrane, and of protoplasm. In the nucleus are fluid and a meshwork of threads, or chromatin. In the protoplasm, alongside of the nucleus, is a small body, known as the centrosome. When a cell is about to divide, the centrosome first divides, and the bodies which are thus formed become arranged at the opposite poles of the nucleus. About these "polar bodies" is seen a peculiar radiate appearance, known as the "spindle." The chromatin of the nucleus then becomes massed in thick threads, and this thick thread then separates into fragments to form loops, which usually are arranged with the open ends of the loops directed toward the periphery, while their apices are at the equator of the nucleus. These loops divide longitudinally, and one-half becomes arranged about each centrosome, thus forming two "daughter nuclei." This division of the nucleus is followed by a constriction of the protoplasm, which divides into two parts, one about each nucleus.

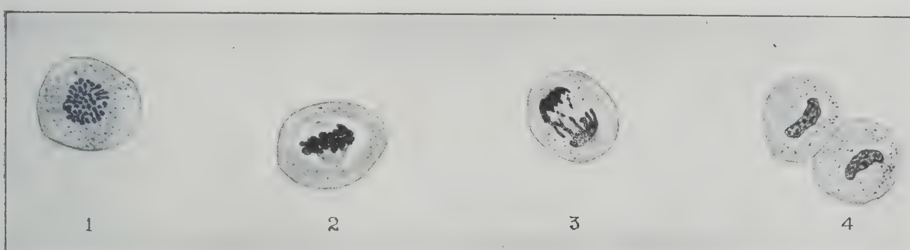

Fig. 74.—Mitosis of Cells, from Human Cancer. 1, Thickening of nuclear chromatin, with disappearance of nuclear membrane; 2, spindle stage, equatorial arrangement of chromatin; 3, later spindle stage, chromatin becoming arranged about the poles of the spindle; 4, division of cell. (*Original.*)

Comparative Power of Regeneration of Different Tissues.

The comparative power of regeneration varies greatly in different tissues.

1. Epithelium, both of the skin and of the internal organs, has great power of regeneration, but the degree to which the epithelium of different organs can regenerate varies greatly. In all cases the regenerated epithelium arises by mitosis from similar adjacent epithelium.

In the skin, after a loss of surface epithelium, new epithelium grows over the denuded surface from the epithelial cells at the edge, and also may arise in the centre of the denuded area, if any islands of epithelial cells are left in the base of the ulcer. Also, epithelial cells will arise from bits of epithelium transplanted

from distant parts of the same animal or of another animal of the same species. In human beings advantage is taken of this latter fact in cases of extensive burns or abrasions by transplanting small bits of epithelium to the denuded surface, giving numerous foci from which new epithelium may arise. Of recent years a better method has been employed, the so-called "Thiersch method" of skin grafting. By this method large pieces of epithelium are taken from the sound skin and transplanted to the new area, the pieces being fitted in such a way as entirely to cover in the ulcer. The deeper layers of this epithelium become adherent to the denuded surface, and in a very brief time the defect is entirely closed by the transplanted epithelium and by epithelium arising from the edges of the transplanted pieces. The sound area from which the epithelium is removed is also very quickly covered in by epithelium, for, since the entire thickness of epithelium is not removed in the transplanted piece, the epithelial cells which are left scattered over a broad area quickly proliferate and form new epithelial cells to replace those which have been removed.

In the uterus there is marked destruction of uterine lining epithelium at the time of menstruation and pregnancy, but the cells are very rapidly reproduced from the epithelial cells of the uterine glands. The same thing happens after very extensive removal of uterine epithelium by curetting.

In the intestine the epithelium has great power of regeneration. An ulcer of the intestine, as in typhoid, is rapidly covered in by epithelium growing in from the sides, and even imperfect crypts of Lieberkühn are produced, although villi are not formed. In wounds of the intestine the inturned edges ulcerate, and are rapidly covered over by epithelium in the same way, so that in a very short time (two or three weeks) it may be impossible to detect the line of the wound.

Liver epithelium has considerable power of regeneration. It has been shown by Ponfick that large areas of the liver of animals may be removed and a new formation of liver substance may take place. When small areas of liver epithelium are destroyed, the lost cells may be replaced by a new formation of cells at the periphery of the lobule—*i.e.*, through proliferation not only of the cells adjacent to those lost, but even of those at a considerable distance.

Kidney epithelium has a very limited power of repair. If single cells of the kidney tubules are destroyed, they are replaced by proliferation of adjacent epithelial cells; but if any considerable amount of renal tissue is destroyed, to supply which new renal tubules must be formed, the lost epithelium is not regenerated, but only scar tissue is produced. Destroyed glomeruli are not reproduced.

2. Mesenchymal tissues. In tissues of mesenchymal origin, connective tissue, cartilage, bone, etc., lost tissue is replaced by tissue similar to that destroyed. But in these closely allied tissues a certain amount of exchange of function may take place after a loss of tissue; *e.g.*, a loss of substance in cartilage, which has a very limited power of regeneration, is usually replaced by con-

nective tissue, although in time this connective tissue may be converted into cartilage and complete regeneration take place.

In bone, after a loss of tissue, *e.g.*, after a fracture, there may be a new formation of tissue, which may in the course of time lead to complete regeneration of the bone. This theoretically possible regeneration is but seldom seen to any great extent in human fractures.

In muscle, after a loss of substance, *e.g.*, after section or rupture, the muscle fibres do not regenerate, although peculiar changes take place in the cut muscle ends which have been described as regeneration, but which really represent degenerative changes. The resulting defect in muscle is filled by a new formation of scar tissue derived from the connective tissue of the muscle sheaths.

II. REPAIR.

General Remarks.

By repair of a tissue is meant the process by which an injury to, or a loss of substance of, the tissue is made good. This, however, does not imply that the lost tissue is replaced by tissue exactly of the same character, or with the same function as that of the original tissue. In fact, so far as the surgeon is concerned, as a rule the loss in specialized tissues is replaced entirely by connective tissue, arising from pre-existing connective tissue, which finally becomes dense connective tissue or scar.

Resolution.—As has been shown in the article on inflammation (*q.v.*), after any injury to any tissue of the body there occur a series of changes in the tissues about the injured point, confined at first almost entirely to the blood-vessels of the adjacent tissues. As a result of these changes there is an escape of the contents of the blood-vessels, *i.e.*, serum, leucocytes, and red blood corpuscles, with a formation of fibrin from the serum, so that the tissues about the injured area are infiltrated with the material which has escaped from the vessels, *i.e.*, the tissues about the injured area are filled with an "inflammatory exudate." In certain cases, usually when the injury has been a slight one and the amount of exudation is small, the exudation may be removed with no further changes in the tissues, and the function of the tissues is renewed. This simple process is spoken of as *resolution*.

Removal of Exudation.—The exudation lies in the interstices of the tissues, and consists of serum, leucocytes, and fibrin, with perhaps red blood corpuscles. The fluid serum is carried off chiefly through the lymphatic vessels, as is shown by the fact that the lymphatics surrounding such an area are dilated in microscopic sections; and if the injury is produced in an extremity, the lymphatics leading from the limbs show an increased flow of lymph. Some of the serum may, however, be taken up directly by the blood-vessels.

The leucocytes in the exudation also are partly removed by lymphatic vessels or by blood-vessels, but a large proportion are destroyed by newly formed endothelial cells, which arise chiefly by mitosis of the endothelial cells of the lymph spaces in the vicinity of the injured area. These newly formed cells take up and include the leucocytes, and destroy them by a kind of digestion. This process is called phagocytosis. If the exudation contains a large proportion of leucocytes, *i.e.*, if the exudation is a purulent one, the removal of the leucocytes is more difficult. In some cases the fluid portion of the exudate is removed by the blood-vessels, while the remaining mass of leucocytes undergo fatty degeneration and necrosis, and are converted into a soft, greasy mass, which may be surrounded by a layer of dense scar tissue, thus being encapsulated. Such a mass may persist in the tissues for a long time. In some cases lime salts may be deposited in such an area, which thus becomes gritty, or even may become completely calcified, and may persist as a calcified foreign body for a long period.

More commonly, in purulent exudations which lie near the surface of the body, or near hollow viscera, the process of exudation continues until the tissues which surround the purulent exudate become necrotic and soften, and finally the surface is reached and the exudation is spontaneously evacuated upon the surface, or into a hollow viscus. In this way most of the leucocytes are evacuated. In the surgical treatment of purulent exudations (abscesses), the surgeon expedites the removal of leucocytes by making artificial openings with the knife. In this way much destruction of tissue is avoided, and in many cases the opening is better placed to allow complete removal of the exudate.

Fibrin, when in small amounts in the tissues, undergoes certain changes, swelling up and softening and undergoing a sort of digestion in the fluids, and then being absorbed by the lymphatics. At times, when fibrin forms large masses in the tissues, it may be partly removed by phagocytic action of giant cells. When fibrin forms on surfaces connected with the outside of the body, it may be removed in considerable masses or as plugs or casts. An open ulcer may be covered by a crust or scab, largely fibrinous, which finally is separated by the growth of epidermis beneath it. In pneumonia much of the fibrin is expectorated as fibrinous plugs. When fibrin forms upon a surface which is not connected with the surface of the body, as in the pleural cavity or on the surface of the peritoneum, it is replaced by granulation tissue, composed of newly formed connective tissue and new blood-vessels, which grows into, softens down, and removes the fibrin. This granulation tissue becomes dense scar tissue finally, and thus produces thickening of the surface on which the fibrinous exudate appeared; or, if the fibrin was on two adjacent surfaces, there will result adhesions composed of dense scar tissue.

The red blood corpuscles of the exudation, if present in small amounts, may be carried off in the lymphatic vessels. If they are present in great quantity some of the corpuscles remain in the tissues, and in that case the hæmoglobin of

the corpuscles becomes dissolved out and forms pigments which produce the color seen in the "black-and-blue" spots. The framework of the corpuscles finally is dissolved in the fluids of the tissue, while the pigment is taken up by phagocytic cells.

Repair.—As has been stated already, after any injury of any tissue of the body, of such a nature as to cause a destruction of tissue, the injury is immediately followed by changes confined to the blood-vessels—changes which lead to the formation of an inflammatory exudation. Within a very short time, twenty-four hours at most, further changes occur, both in the cells of the tissues and in the blood-vessels surrounding the point of injury, which lead to the formation of new tissue to replace the lost tissue. In these changes new cells are formed by mitosis of adjacent cells, and new blood-vessels are formed by outgrowths from adjacent blood-vessels. This formation of new cells and new blood-vessels to replace destroyed tissue is described as the process of repair. The process of exudation and the process of repair, however, are not sharply separated, as the exudation persists for some time after the process of repair has begun. Ultimately, the exudation disappears and is replaced by new cells and blood-vessels, but for a considerable time inflammatory exudation and cells and vessels exist together. After the exudation disappears, the newly formed tissue undergoes a series of changes which convert the cellular vascular tissue into a dense scar.

Processes of Repair in Different Tissues and Organs of the Body.

A. Repair of Wounds in Soft Tissues.

In every wound of the soft tissues the immediate result of the injury is destruction of tissue. This destruction of tissue is immediately followed by the appearance of an inflammatory exudation about the point of injury. In wounds which heal without becoming infected by pathogenic micro-organisms (aseptic wounds) the amount of exudation is directly proportionate to the extent of the wound. In wounds which become infected with micro-organisms (septic wounds) the amount of the exudate is dependent upon the extent of the wound and upon the amount of injury due to the action of the toxin produced by the micro-organisms. Within a few hours (twenty-four hours at most) after the appearance of the exudation, there begins a new formation of cells and blood-vessels to replace the lost tissue. The general steps of the process are the same whether the edges of the wound can be approximated (incised or closed wound) or cannot be brought together on account of extensive destruction of tissue (lacerated, contused, or open wounds); but there are slight differences in the details of the process, as well as in its duration, so that it is customary to describe the healing of aseptic closed, and open wounds separately.

Repair of Aseptic Closed Wounds; "First Intention."—The histological de-

tails are seen best in experimental wounds on animals. The cellular changes are best studied in a non-vascular organ like the cornea; the vascular changes, in wounds in a relatively simple tissue, such as the ear or tongue of a rabbit.

FIG. 75.—Repair of Wounds; First Intention. Experimental incision in cornea of rabbit. Condition after the lapse of two days. The picture illustrates the new formation of connective-tissue cells in a non-vascular organ. 1, Newly formed epithelium growing across the line of incision; 2, line of incision filled with exudate, leucocytes, and fibrin; 3, 3, 3, early stages of proliferating corneal corpuscles. (*Original.*)

The immediate result of the injury is hemorrhage. In a few hours more the space between the approximated edges is filled with an inflammatory exudate of leucocytes, serum, fibrin, and red blood corpuscles. The exudate extends laterally for some distance into the adjacent tissues, and on the surface coagulates into a thin film or crust (scab). The adjacent blood-vessels become dilated and contain many leucocytes. (Fig. 75.)

In a few hours a new formation ("proliferation") of the epithelial and connective-tissue cells adjacent to the wound begins. Mitotic figures appear in the epithelium, and the epithelium begins to extend over the surface of the wound, beneath the crust, in a thin layer. Mitotic figures (see Fig. 74) appear in the adjacent connective-tissue cells, and young connective-tissue cells, oval or polygonal in shape, are formed, and extend into the exudate from either side of the wound. These new connective-tissue cells at first bear no resemblance to the connective-tissue cells from which they are derived. The new cells are produced in excessive amounts, so that they are at first more than sufficient to replace the lost tissue. While this new formation of cells is going on, a new formation of young blood-vessels also occurs. These new vessels arise from existing blood-vessels, either small veins or capillaries. The endothelial cells of these capillaries enlarge, send out long processes, and undergo mitosis, thus forming long, pointed processes of cells, which finally separate and are arranged as blood-vessels, that extend into the exudate. Some of the processes unite with other similar processes from the same or from the opposite side of the wound, so as to form loops of new capillaries. (Fig. 76.)

This process of proliferation of connective tissue and new formation of blood-vessels continues for a variable time, depending upon the size of the wound, the

character of the tissue, the amount of hemorrhage, and the accuracy of approximation of the edges of the wound. This combination of young connective tissue and new blood-vessels is called "granulation tissue." As the granulation tissue extends into the exudate, the exudate is removed in the manner already described, and finally disappears. Meanwhile the epidermis completely covers in the surface of the wound, becomes thickened, and forms imperfect skin papillæ. The time required to cover in and remove the exudate and replace lost tissue is variable, depending upon the character and size of the wound, but averages from about seven to ten days. (Fig. 77.)

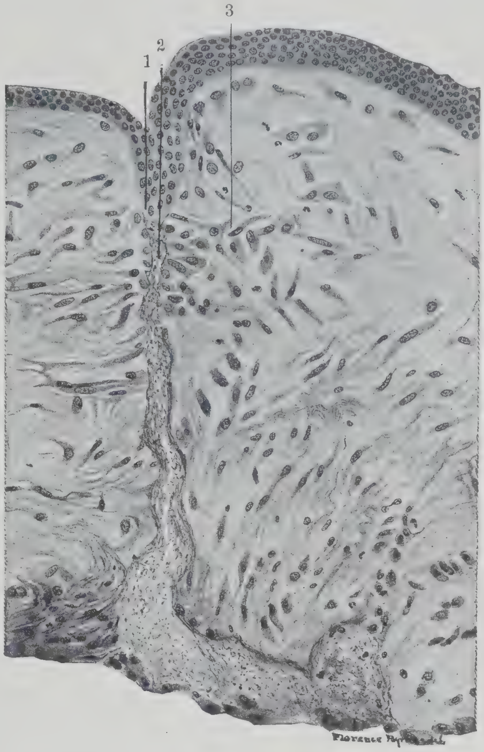

Fig. 76.—Repair of Wounds; First Intention. Experimental incision in cornea of rabbit. Condition after the lapse of four days. 1, New epithelium which has closed the incision; 2, remnant of exudate in the incision; 3, marked proliferation of corneal corpuscles. (*Original.*)

The connective-tissue cells at first are oval or polygonal, but in a few days they become elongated and spindle-shaped, and much intercellular fibrillar material appears between them; and, finally, the nuclei become less numerous and small, as in normal connective tissue, and the intercellular material becomes dense. Many of the young blood-vessels, at first very numerous, disappear, and the remaining tissue shows very little vascularity. Thus is formed dense "scar tissue," which is contractile and occupies much less space than the granulation tissue. This entire process of scar formation takes approximately from ten days to two weeks. The scar still continues to contract and become less vascular, however, for a long period—for weeks and often for months. (Fig. 78.)

In human wounds the process is the same as in experimental wounds, although on account of the extent of the wound and the complex anatomy the process appears more confused. The edges of human wounds practically never are exactly approximated, and, as a result, the interval to be covered by the proliferating epithelium is greater than it is in experimental wounds, and the time required to cover in the exudation is correspondingly increased. The walls of the wound seldom are as distinct and sharply defined microscopically as in the case of experimental wounds, and the exudation extends laterally much far-

ther beyond the line of incision than in the case of experimental wounds. This probably is due, in part at least, to the fact that in surgical wounds the edges undergo a considerable amount of injury from manipulation, thus causing damage far outside the mere line of incision. Consequently, when proliferation of cells and formation of granulation tissue begin, these processes are not confined so closely to the line of the incision as in experimental wounds, but oftentimes extend several centimetres on either side of the incision. Moreover, in human wounds different layers of connective tissue, fascia, subcutaneous fat,

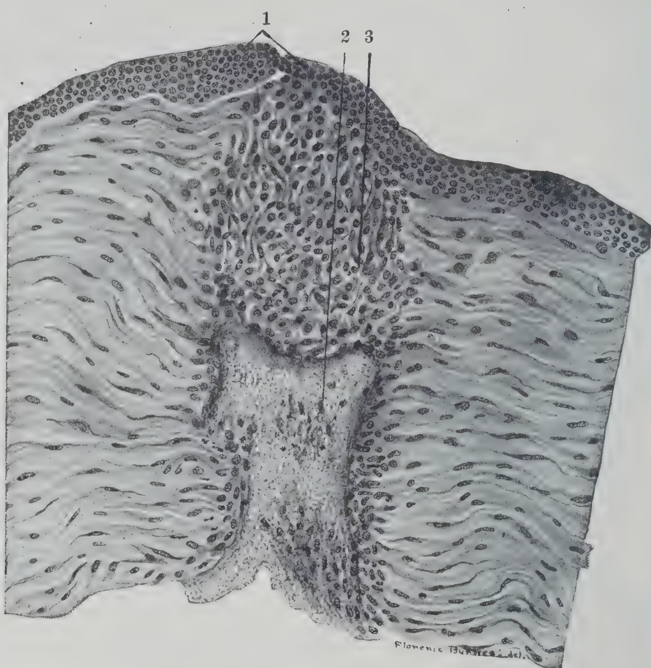

Fig. 77.—Repair of Wounds; First Intention. Experimental incision in cornea of rabbit. Condition after the lapse of six days. 1, New epithelium covering in incision; 2, remnant of exudate; 3, newly formed connective tissue, derived from proliferating corneal corpuscles. (*Original.*)

loose connective tissue, etc., are injured, and the rapidity of growth of the different layers varies considerably. The process, as a rule, takes longer than in experimental wounds, and after a few days lymphoid and plasma cells are seen in the tissues. Also, since the deeper layers of the wounds seldom are exactly approximated, the amount of exudate between the walls of the wound is different in different places. Then, again, since the greater the amount of exudate the greater the length of time required to replace it by granulation tissue, the extent to which organization is completed may vary in different parts of the same wound. The time required entirely to replace exudate by granulation tissue ("organization") amounts, under the most favorable circumstances, to ten days, and, even in perfectly aseptic wounds, several days longer may be required. It should be borne in mind also that, even if the exudate is entirely or-

ganized, granulation tissue in the early stages contains relatively little intercellular fibrillar material, and is therefore inelastic, weak, and unable to stand much strain. (Fig. 79.)

After organization is completed the intercellular material continues to form

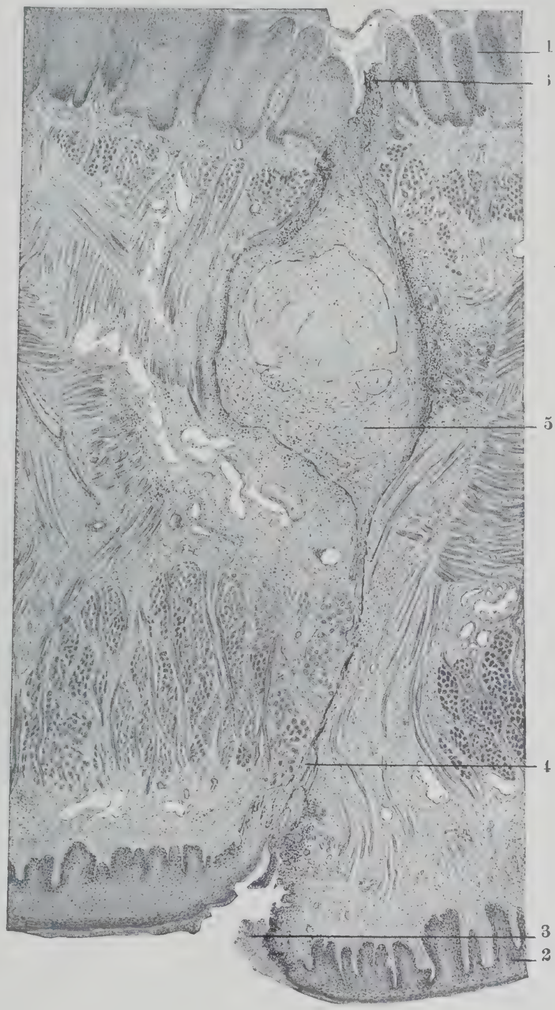

Fig. 78.—Repair of Wounds; First Intention. Experimental incised wound through tongue of rabbit. Condition after the lapse of twenty-four hours. 1, Epithelium on dorsum of tongue; 2, epithelium on bottom of tongue; 3, exudate of leucocytes and fibrin at surface of wound; 4, line of incision filled with exudate, chiefly fibrinous; 5, gap in wound, caused by retraction of muscle fibres, filled with exudate of fibrin, serum, and relatively few leucocytes; 6, exudate of leucocytes and fibrin. 3 and 6 correspond to the crust seen clinically in wounds, but, on account of the moisture of the mouth, it is less marked than usual. (*Original.*)

and contract, while the blood-vessels of the granulation tissue disappear until a very dense, white scar tissue is left.

The gross appearances in human wounds correspond to the histological

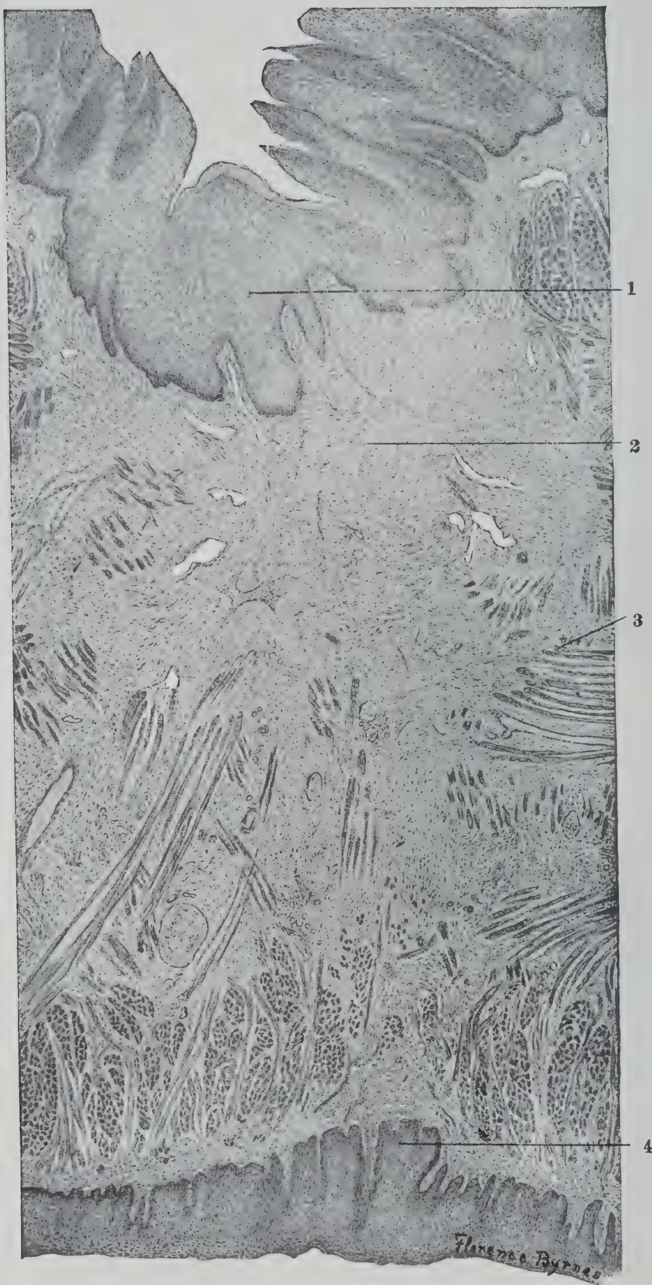

Fig. 79.—Repair of Wounds; First Intention. (Experimental.) Incised wound through tongue of rabbit. Condition after the lapse of eight days. Line of incision is not clearly defined, owing to the fact that new connective tissue has formed beyond the limits of the original incision. 1, Newly formed epithelium at the point of incision, papillæ irregular; 2, young connective or granulation tissue, with fewer blood-vessels than in preceding section; 3, club-shaped degenerated ends of muscle fibres included in the young scar; 4, new epithelium closing in line of incision. (*Original.*)

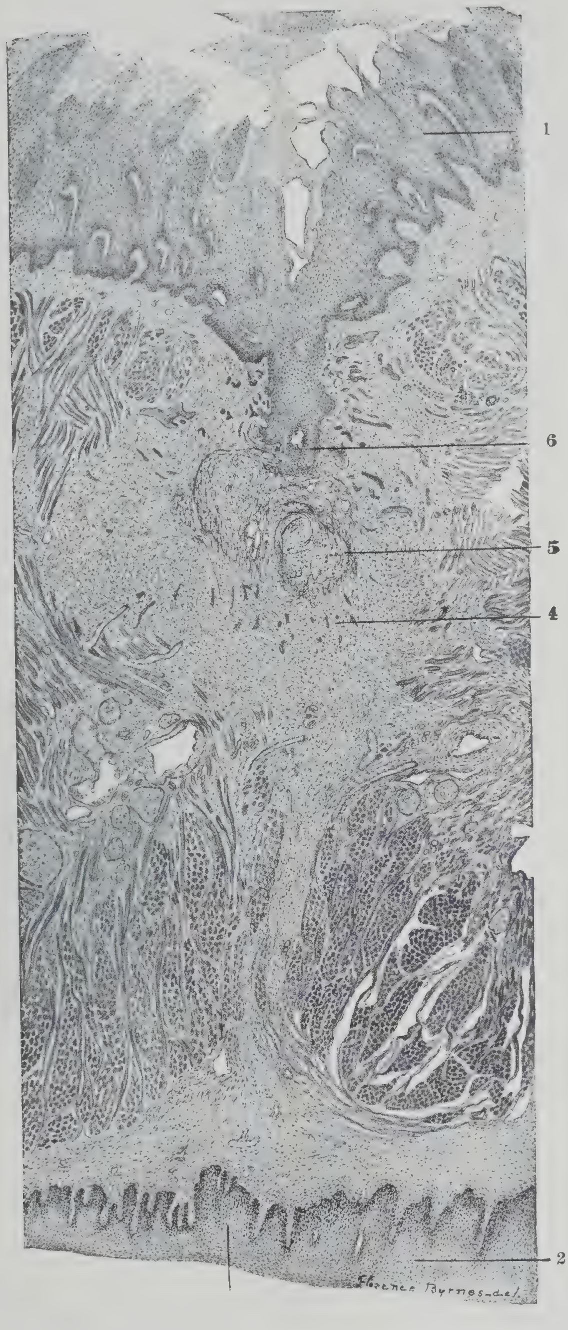

Fig. 80.—Repair of Wounds; First Intention. Experimental incised wound through tongue of rabbit. Condition after the lapse of five days. 1, Dorsum; 2, bottom of the tongue; 3, ingrowth of epithelium from edges of wound, closing surface of incision; 4, remnant of exudate along line of incision; 5, newly-formed granulation tissue, arising from adjacent connective tissue, and blood-vessels, extending into and replacing exudate; 6, point of incision closed by newly-formed epithelium, which forms nearly normal papillæ. Wound is wider on top than below on account of retraction of muscle fibres. (*Original.*)

changes just described. At the end of a few hours after an incision has been made the edges of the wound are sealed together by an adhesive layer of exudate, slightly yellow if hæmostasis has been complete, or tinged with red if hemorrhage has occurred. In from twenty-four to forty-eight hours the surface of this exudate becomes hard and dry, and forms a crust or scab, while the edges are infiltrated with exudate, and the old blood-vessels are somewhat dilated. A certain amount of redness of the edges always appears, even when the wound is perfectly aseptic. The edges, too, are somewhat hot and tender. If the edges of the wound are torn apart at this time, but little hemorrhage takes place, as no new vessels are yet formed, and the walls of the wound look opaque and gelatinous from the presence of exudate. If the crust is removed in from three to six days, the surface of the wound is seen to be covered with a thin, pearly layer of proliferated epithelium. If at this time the edges are separated it will be found that this can be done only with some difficulty and that the raw surfaces bleed freely, on account of injury to the newly formed vessels of the granulation tissue. After about the tenth day the crust separates spontaneously from the underlying layer of newly formed epidermis, which is whiter and thicker than at first, while the line of incision is filled with vascular red scar tissue, composed of spindle-celled young connective tissue in which are numerous young blood-vessels. In the course of weeks or months the scar becomes white and narrower than it is during the red-scar stage, on account of the disappearance of the red blood-vessels and the conversion of the young connective tissue into dense, contractile connective tissue. (Fig. 80.)

Repair of Aseptic Open Wounds or Ulcers; "Second Intention," "Healing by Granulation."—As the result of injury there may take place a more or less extensive destruction of the superficial and underlying tissues of the body, producing a wound so extensive that the edges of the skin cannot be approximated so as to close it. In such cases the details of the reparative process differ from the process which takes place in closed wounds, although in their general features the two are alike.

In such an injury of the surface of the body the hemorrhage is usually somewhat copious, although, if the wound is very superficial, the loss of blood may be slight. The blood which thus escapes may ultimately coagulate upon the surface of the wound to form a red scab. In any case, in a few hours there is formed upon the surface an exudate, which consists of fibrin, leucocytes, and serum; and this exudate also extends to a considerable distance into the tissues about the wound. Adjacent blood-vessels are moderately distended. In a few hours, by the formation of fibrin and the coagulation of blood, the wound is covered with a crust or scab.

Very shortly (twenty-four hours) a proliferation of epithelium at the edges of the wound, a new formation of blood-vessels from pre-existing capillaries and veins, and a proliferation of connective-tissue cells from the connective tissue

about the wound take place, just as in the closed wounds. The epithelium grows into the exudate on the surface as a thin, tongue-like film, but epithelium has a very limited power of growth into exudate, and, if the denuded surface is extensive, may be unable to cover the surface of such an open wound for many

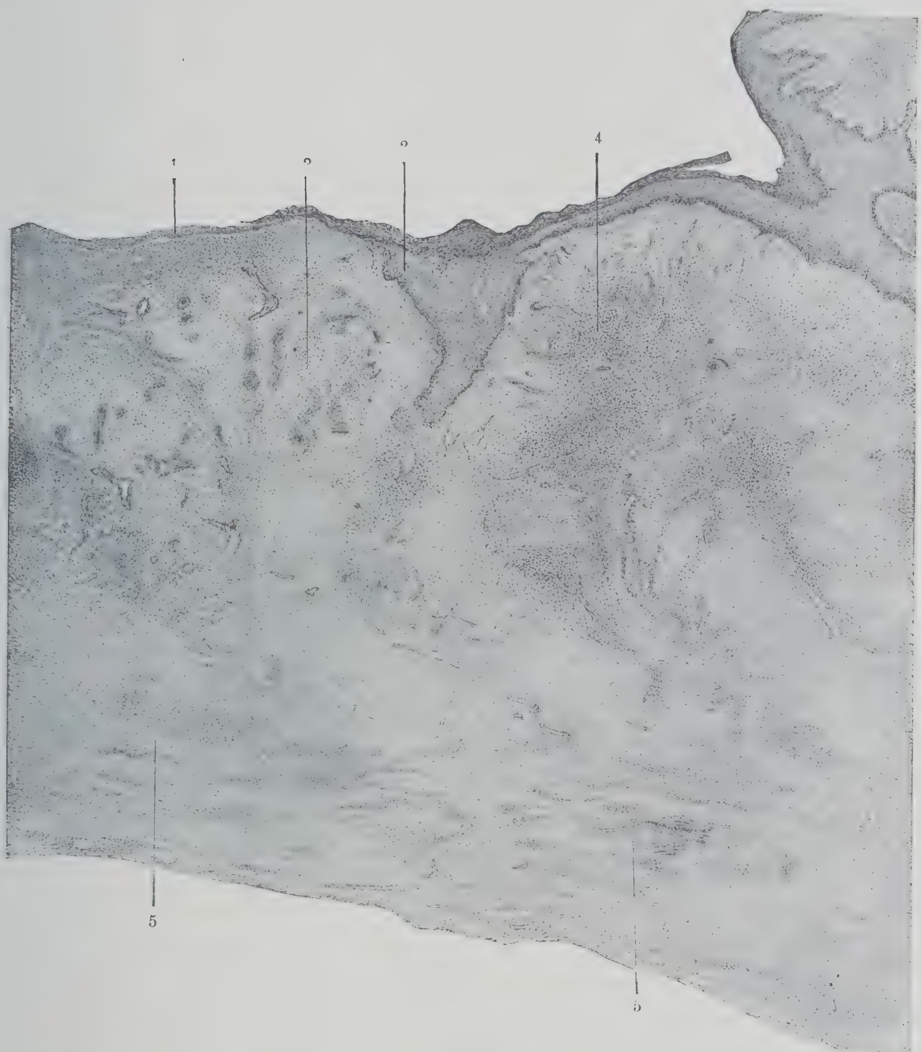

Fig. 81.—Repair of Wounds; Granulation. Human wound, of several months' duration. 1, Fibrinous exudate on surface of ulcer; 2, epithelium extending over surface of granulations; 3, granulation tissue, beneath exudate; 4, granulation tissue which has been covered by ingrowth of epithelium; 5, 5, base of dense fibrous tissue, formerly granulation tissue. (*Original.*)

weeks or months. If the wound remains open the epithelium may form a thickened, rounded edge, dipping down into the underlying granulation tissue.

By proliferation of the adjacent connective tissue young connective-tissue cells, at first polygonal or oval, later spindle-shaped, and finally fibrillated, are formed and extend into the exudate from the sides and from below. Accom-

panying these cells are newly formed vessels, appearing as pointed processes arising from the endothelium of existing blood-vessels. Ultimately, these processes divide and form hollow tubes or blood-vessels. Adjacent tubes meet and unite, thus forming vascular loops. This loop formation gives to that portion of the wound uncovered with epithelium a granular appearance; hence the term "granulation tissue." The blood-vessels in the granulation tissue are very abundant. From this surface of granulation tissue an inflammatory exudate is given off. Besides the leucocytes many lymphoid and plasma cells are seen in the granulation tissue. Both lymphoid and plasma cells are destroyed in part by the phagocytic cells of the granulation tissue. The latter tissue grows until it reaches the level of the surrounding epidermis, and, if the wound is not too large, it ultimately becomes covered by epidermis. In many cases the wound is so large that a very long period is required for the accomplishment of this. In such cases granulation tissue grows above the level of the skin, and forms exuberant granulations, or "proud flesh." This often is seen in wounds of large size. In small wounds the process of repair may be completed in a few days (about fourteen). The fibrillation of the connective tissue begins early in the deeper layers, while the superficial layer continues to be composed of polygonal or oval young cells. In extensive wounds, which are uncovered by epithelium after months, the deep layers of the ulcer are composed of dense scar tissue, whose fibres run mostly parallel with the base of the ulcer.

The gross appearances correspond to the histological changes, and are seen, *e.g.*, in slightly "barked" knuckles. In that case the wounded surface is covered in a few hours with a clear yellow fluid, which coagulates in a short time and forms a thin film or crust. If the wound is deeper, hemorrhage occurs and the crust is red from coagulated blood. In two or three days, if the crust is removed, a thin, pearly film of proliferating epithelium is seen advancing beneath the edge of the crust. The uncovered portion of the wound shows a red, bleeding, granular surface of granulation tissue. If the wound heals promptly the epithelium covers the entire surface in the form of a thin film, the underlying granulation tissue becomes fibrillated, and forms a moderately elevated, firm, red scar. After weeks or months this red scar disappears. As the vessels disappear the scar contracts and becomes markedly smaller than at first, and white. (Fig. 81.)

If the wound is extremely large or the process of healing for any reason delayed, the wound may fail to be covered by epithelium for weeks or months. In that case the edges of the wound are covered with a zone of epithelium of variable width, often very thick and depressed at the edge, which advances very slowly. The rest of the wound is filled by reddish, granular, moist, vascular granulation tissue, which bleeds easily and often extends above the level of the epithelium at the edge. If this layer be scraped off, the base of the ulcer is seen to consist of very dense, white scar tissue. This dense tissue makes the best base for the application of skin grafts, because there is less or no inflam-

matory exudate in this dense layer, and there is less tendency to float off the grafts by exudation. (Fig. 82.)

Repair by "Third Intention."—Occasionally in wounds in which there has been a considerable loss of tissue, it is possible, after a few days, to approximate the edges of the wound in such a way as to convert what was originally an open wound into a closed one. In such cases the early stages of repair are like those of the open wounds, *i.e.*, a proliferation of epithelium at the edges and a formation of granulation tissue at the bottom of the wound, take place. If the edges then are closed by pressure or by approximation sutures, surfaces of granulation tissue, covered with a varying amount of exudate, are approximated in the deeper part of the wound, while, at the surface, edges of proliferating epithelium are brought together. In such cases, under favorable circumstances, the open wound is converted into a closed one, the granulation tissue from either side grows into and organizes the exudate, the epithelium grows over and closes in the wound, and the later stages of repair are like those of a wound which is a closed one from the beginning.

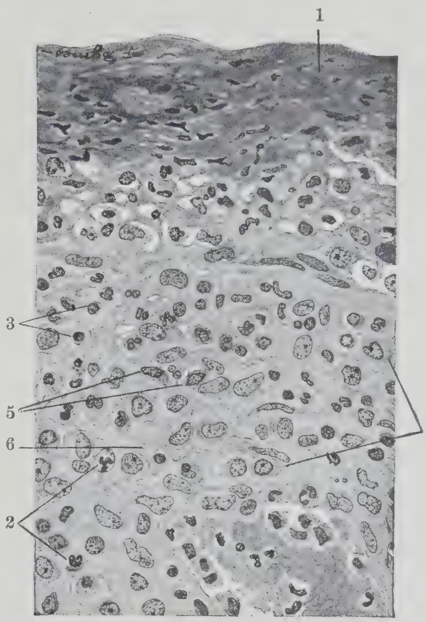

FIG. 82.—Repair of Open Wounds; High Power Details of Surface of a Granulating Wound (see Fig. 81). 1, Fibrinous exudate on surface, enclosing partly disorganized leucocytes; 2, leucocytes; 3, lymphoid cells; 4, plasma cells; 5, fibroblasts, or newly formed connective-tissue cells; 6, new intercellular fibrils. (*Original.*)

Infected wounds. In the wounds which become infected by pyogenic micro-organisms there is danger of general infection or of thrombosis and embolism, while the general character of the local wound itself may be altered, the area of the wound enlarged, the process of repair modified, and the time required for complete repair prolonged.

The pyogenic organisms commonly present in infected wounds produce practically one of two types of lesion—*i.e.*, they produce a marked solution of tissue, such as is seen in the ordinary abscess due to infection by Staphylococcus pyogenes aureus; or they produce a diffuse necrosis of tissue, such as is seen in the phlegmonous inflammation produced by the streptococcus. The effect produced upon infected wounds varies with the type of infection present.

In closed wounds in which the infection is of the dissolving type the first effect of infection is to produce an increase in the amount of inflammatory exudate present between the edges of the wound, and this increased exudate consists almost entirely of leucocytes, instead of much fibrin with relatively few

leucocytes, as is the case in aseptic wounds. The infectious organisms also extend rapidly into the tissue at the edges of the wounds, cause a marked solution of those tissues, and enlarge the interval between the edges. If the infection occurs early the exudate may be discharged between the sutures, but if it occurs after the edges are sealed together by fibrinous exudate the wound rapidly is converted into an abscess cavity, which ultimately may open upon the surface at some point in the original incision; or the entire margin of the wound, including the epidermis, may become dissolved, and the closed wound may be converted into an open wound. In either case the extent of the wound is very much enlarged, and the amount of tissue to be replaced is much increased and the time required for repair correspondingly prolonged.

In case the closed wound becomes infected by organisms which produce necrosis without marked solution of tissue, *i.e.*, an acute inflammation of the phlegmonous type, the process of repair also is affected. In such cases the pyogenic organisms extend into the lymphatic clefts of the tissue adjacent to the wound and produce necrosis of tissue, and the necrotic area becomes infiltrated with a purulent exudate. The necrosis may extend over an area many times greater than that of the original wound. After the infection has ceased or the wound has been artificially drained, a large slough, usually but not always subcutaneous, is formed about the wound, and this necrotic tissue must be replaced by granulation tissue, the time required for this replacement corresponding to the extent of the necrosis. In such cases the area of granulation tissue is many times greater than that which would be inferred from the line of the original incision, and may, as in the case of a limb, lead to the formation of a very extensive subcutaneous scar, which may surround the entire limb as a buskin of scar tissue, which, by its pressure and contractility, may lead to very great impairment of the functions of underlying muscles.

In open wounds the result of an infection is similar to that in closed wounds. An open wound infected with organisms which produce suppuration and solution of tissue may have its original area enormously increased, while the time required for healing is correspondingly lengthened. An open wound infected with organisms which produce phlegmonous inflammation not only has its superficial area increased to a considerable extent, but also becomes surrounded by a subcutaneous slough which leads to the same complications as are seen in infected open wounds.

Principles of Treatment of Wounds.

From a consideration of the process of repair of wounds certain simple fundamental principles of treatment are obvious.

Surgical cleanliness is the most important factor, and is practically under control. This cleanliness applies to the field of operation, to the hands of the operator, instruments, sponges, sutures, dressing, and to all materials which in

any way are brought into contact with the wound. If perfect surgical cleanliness (asepsis) is obtained, the amount of tissue to be repaired is dependent solely upon the size of the original wound. If pyogenic infection occurs the destruction of tissue depends upon the extent of infection, and in all cases the extent of the wound and the length of time required to replace the defect are increased, to say nothing of the dangers of septicæmia, etc.

Avoidance of manipulation is also extremely desirable. In wounds in which long-continued or violent manipulation is carried on, the destruction of tissue extends very widely beyond the mere limits of a surgeon's incision, and in such cases the amount of tissue to be replaced is much greater than the mere incision would require. Even in incised wounds the extent of the reparative process beyond the line of incision is much greater than usually is appreciated. For this reason it is desirable for the surgeon to make free, sweeping incisions, rather than a series of little cuts.

Perfect and complete hæmostasis also is necessary to obtain rapid healing of wounds. The greater the amount of hemorrhage between the edges of aseptic wounds the greater the length of time required for healing. The hemorrhage separates the edges of the wound and increases the area to be organized by granulation tissue. Excessive amount of blood in a closed wound also furnishes an excellent culture medium for the growth of pyogenic organisms, if any are present.

Accurate closure and approximation of the edges of wounds in which an attempt is made to obtain primary union are essential. The approximation should affect not only the superficial edges, but especially the deeper layers of the wound. Even in the case of careful operators it is astonishing to see, on examining sections with a microscope, how very imperfect the closure of the wound is. The more accurately the epidermis is approximated the less the surface to be covered by proliferating epithelium. The quicker the wound is covered by epithelium the less the liability of infection. The more carefully the deeper layers are approximated and dead spaces are obliterated the less the amount of inflammatory exudate and blood to be removed and organized by granulation tissue, and the smaller the scar.

Aseptic protection of the wound is essential during the early days of the reparative process. The danger of secondary infection is over when the wound is covered by epithelium and the exudate is entirely replaced by granulation tissue. The power of resistance to infection possessed by a wound covered with granulation tissue is much greater than that which it possesses in the earlier stages before the formation of granulation tissue.

Fixation of wounded tissue also is essential if it be desired to protect fresh or granulating edges of the wound from further injury. This fixation may be obtained in a variety of ways.

In regard to open wounds there are certain special precautions. Surgical asepsis is as desirable in them as in closed wounds, but perfect asepsis is not

feasible in wounds which remain open for long intervals. The reason why extensive open wounds do not oftener become seriously infected is that healthy granulation tissue has a marked power of resistance to absorption of pyogenic organisms. On the surface of open wounds, in the early stages, masses of necrotic tissue ("sloughs") often are present. It is better not to attempt too vigorous removal of these, as their forcible removal leads to repeated traumatism of the young granulation tissue beneath them, with a consequent prolongation of the time of healing and increased danger of pyogenic infection.

In open wounds also it is desirable to keep the granulation tissue below the level of the advancing epithelial edge, as epithelium often is unable to cover over exuberant granulations.

In many cases of extensive open wounds the epithelium ceases to advance over the granulating area, and in such cases it becomes necessary to cover in the epithelial defect by small isolated grafts, plastic flaps, or Thiersch grafts.

Regulation of the blood supply always is desirable in open wounds. Venous stasis always appears to interfere both with the formation of granulation tissue and with the advance of the epithelium. Prevention of venous stasis can be obtained by pressure, by removal of varicose veins, or by position.

B. Sutures and Other Foreign Bodies.

Various substances are used for approximating, supporting, and holding in position the edges of wounds. These mechanical supports must be retained in the tissue until the process of repair along the line of incision is so advanced that the new tissue can support the tension upon the wounded area. Usually, when the wound is superficial the ends of the suture are left visible, and the suture is removed when the repair of the wound is sufficiently advanced, although occasionally even skin sutures are buried. In deep wounds or in wounds of the various body cavities, sutures or ligatures may be buried, and cannot be removed after healing is completed.

The material used as sutures may be of animal origin, and therefore capable of being ultimately dissolved by the tissues. Other varieties of sutures cannot be dissolved. Of the soluble sutures those most commonly used are catgut, both plain and chromicized, and various animal tendons. The common insoluble sutures are silk, silkworm gut, horsehair, celloidin, and various metallic wires.

The character of the reaction produced in the tissues by sutures depends partly upon whether the suture is soluble or insoluble. The primary effect of the introduction of a suture is the production of a minute wound, which is filled by a foreign body. The soluble sutures, of which catgut may be taken as a type, at first act as a foreign body, but after a time are dissolved by the tissues, and the gap left by their removal becomes filled with scar tissue. Insoluble sutures, such as silk, persist indefinitely unless removed, and finally are surrounded, infiltrated, and encapsulated by scar tissue.

Soluble sutures. The introduction of the suture produces a minute wound. Along the track of the suture, extending into the tissues for some distance beyond it, and also extending into the clefts of the suture itself, comes an inflammatory exudate. The suture itself becomes swollen and fibrillated, and finally begins to dissolve. By the third day a layer of granulation tissue appears about the suture. In this granulation tissue very few, if any, giant cells are seen. The granulation tissue advances, while the suture disappears, and the exudation is absorbed, until finally no remnant of suture can be seen and the track of the suture is occupied by granulation tissue, which becomes fibrillated and is converted into a white scar.

The length of time required to effect the complete removal of absorbable sutures is variable. Small-sized, plain catgut requires approximately twelve days; larger sizes take somewhat longer. Chromicized catgut takes a variable time, dependent upon the degree of chromization, and in some cases the suture may be rendered practically insoluble and may persist for months or even years. Soluble sutures which become infected by pyogenic organisms are absorbed much less rapidly than sutures which remain aseptic.

Insoluble sutures. As in the case of the soluble sutures, the first effect is the production of a minute wound containing a foreign body. Into this wound comes an inflammatory exudate. The exudate extends for some distance into the surrounding tissues and also into the meshes of the suture. In a few hours granulation tissue is formed at the periphery of the wound and extends toward the suture, and finally the exudate disappears and the granulation tissue surrounds and extends between the fibres of the suture. In this granulation tissue are many giant cells. These giant cells may persist for months or years. In the case of superficial sutures, which are removed at the end of ten or twelve days, there is left a minute wound lined with granulation tissues, and this wound in a very short time is filled with new granulation tissue.

Insoluble sutures, which are fibrillar like silk, are surrounded and everywhere enmeshed by the scar tissue which penetrates between the fibres. Sutures like wire, horsehair, or silkworm gut are not fibrillar, and no enmeshing by the scar tissue takes place.

C. Wounds of the Intestine.

When any portion of the intestinal tract is wounded, it is essential that the wound be closed at once in such a way as to render the wall of the intestine watertight as soon as possible, so as to prevent leakage of the infectious contents. Consequently, many methods have been devised for securing mechanical closure as perfect as possible—it is, however, never absolutely perfect; and the serous surfaces of the cut edges always are approximated, because, if mucous-membrane surfaces are brought together, repair does not begin until the epithelium has been sloughed off; while, when the external (serous) sur-

faces are approximated, the production of fibrinous exudate is very rapid, and in a very few hours the wound is rendered watertight, provided it be not subjected to too much mechanical tension. Many methods of suture have been devised for closing the intestine. In some cases the suture may penetrate all the coats of the gut, but these sutures are, as a rule, applied merely to give fixation of the wounded edges. The sutures which approximate the serous surfaces of the intestine should not extend from the lumen of the intestine to the peritoneal cavity, for if they do they make a wound that is connected with the infected intestinal canal, and infection along the suture may lead to infection of the general peritoneal cavity. The best suture is one which gives the strongest and most perfect immediate mechanical closure of the wound without allowing any connection of infected intestine with the peritoneal cavity, and also gives the most perfect approximation of the edges of the external serous coat without diminution of the calibre of the intestine. It may be said that without doubt the best suture material is, on the whole, silk or celloidin, as animal sutures soften so early that they do not maintain perfect approximation until the wound is completely organized. Mechanical devices should be used only for special clinical reasons. The process of repair is the same, no matter what mechanical method is used, but the process which I have described above is such as is seen after suture.

In intestinal wounds the mucous membrane is inverted and the serous surfaces are approximated. The interval between the approximated serous surfaces thereupon quickly fills with inflammatory exudate, and all layers of the cut intestine are infiltrated. In a few hours the endothelium of the serous membrane becomes necrotic between the sutured edges, as well as for a considerable distance beyond the line of incision, and the latter can no longer be recognized. Exudate covers the external surface of the gut for a considerable distance beyond the wound, and thus in a few hours the wound becomes impermeable to fluids, if too much tension be not applied. The inverted mucous membrane becomes necrotic, and always is more or less infected. Through a process of necrosis the invaginated portion of the gut becomes dissolved. In a very few hours (twenty-four) a marked proliferation of the connective tissue, chiefly of the subserous connective tissue, takes place, together with a new formation of blood-vessels. This granulation tissue very rapidly extends into the exudate between the inverted serous surfaces, and in a relatively short time, often by the seventh day, the exudate is entirely removed and replaced by granulation tissue. While this is taking place, the inverted edges have sloughed, forming an ulcer on the inner surface of the gut, beneath which granulation tissue also forms. The intestinal epithelium at the edges of this ulcer proliferates and extends over the surface of the ulcer, just as does epithelium in ulcers of the surface of the body. While these changes are taking place inside the gut, the exudate on the outside also has been replaced by granulation

tissue. Finally, the granulation tissue between the inverted serous surfaces becomes dense scar tissue, and the internal ulcer is covered by intestinal epithelium, which even may form imperfect glands. In time, the restoration is so complete that it may be impossible to find the site of the wound by gross examination.

The healing usually proceeds more rapidly than it does in wounds of the surface of the body. The wound is sealed by fibrinous exudate within a very few hours, although, of course, the fibrin can easily be displaced under great tension. In many cases the organization of the exudate is completed in seven days, although it is to be remembered that granulation tissue at this time still is very fragile. Rapidity of union is favored by accurate approximation of serous surfaces and by avoiding, as far as possible, any manipulation that might disturb the approximated edges.

D. Repair of Tendons.

Tendons, aponeuroses, and ligaments are special types of connective tissue. What has already been said about the repair of connective tissues in wounds applies in a general way to the repair of tendons, but tendons are connective tissue with a special function and a special structure, and the details of the process of repair, in these structures, vary somewhat from the process as seen in, *e.g.*, subcutaneous connective tissue. It will, therefore, be proper to make special mention of some of these details.

To understand the process of repair it is necessary to bear in mind the anatomy of a normal tendon. Tendons are composed of the densest sort of fibrous tissue arrayed in parallel bundles, closely connected, with relatively few elastic-tissue fibres. Surrounding the tendons is a layer of loose areolar tissue (the peritendineum), from which septa run into the tendon, dividing it into larger (secondary) and smaller (primary) bundles of dense fibres. These dense fibres appear, under the microscope, wavy from contraction, and anastomose more or less with one another. Between the fibres are cells which on long section are oval or rectangular, but on cross section are stellate, and are united to other similar cells by processes, thus separating the fibres into bundles.

When a tendon is divided there always is considerable retraction of the divided ends. This is due partly to contraction of the muscle of the tendon, and partly to contraction of the fibres of the tendon itself. The peritendineum seldom retracts to the same extent as the tendon, but becomes markedly fibrillar and folds over the retracted end of the tendon. Into the interval between the retracted tendon ends comes an inflammatory exudate, with perhaps some hemorrhage. The mesh of the peritendineum is filled with exudate, but this exudate extends only a little way into the cut ends of the tendon itself. Very early there begins a rapid proliferation of connective-tissue cells from the connective tissue of the mesh of the peritendineum, not only between the re-

tracted ends of the tendon, but also from the peritendineum outside the tendon ends, thus forming a spindle-shaped swelling much like the callus of a fracture. At the same time new blood-vessels are formed. In this spindle of granulation tissue intercellular fibrils appear very early to an extent much more marked than in ordinary connective tissue. The cells of the tendon take very little part in this process of proliferation, and the original dense fibres of the tendon not at all. There is, however, marked proliferation of the connective-tissue cells of the connective-tissue septa of the tendon, which extend between the dense fibres of the tendon proper. As the proliferation continues the exudate disappears, and finally the cut ends are joined by a spindle of granulation tissue. The blood-vessels disappear very early, granulation-tissue fibrils are formed in large amounts, and a spindle of dense, fibrous tissue joins the cut ends. In time, the new intercellular fibrils cannot be distinguished from the original tendon fibres, the new tissue becomes of the same size as the original tendon, and cannot be distinguished by the naked eye from the uninjured tendon.

The time required for the process is variable, depending upon the size of the tendon and upon the amount of separation. The formation of completely organized, dense, fibrous tissue in smaller tendons is completed in about two weeks. In larger tendons the process covers a somewhat longer period of time. In practically every case the tendon is sufficiently regenerated to allow passive motion in about three weeks.

In some cases tendons fail to unite or may unite imperfectly. If the cut tendon ends are too widely separated, the connective tissue reproduced by adjacent connective tissue may interpose, and the gap be filled with ordinary scar tissue instead of with connective tissue arising from the peritendineum; or again, even if the two tendinous ends become united with new tendon derived from the peritendineum, the new tissue may become adherent to the new connective tissue of the adjacent skin, etc., and thus imperfect function may result. Also, where several tendons are divided in one wound, *e.g.*, in accidental wounds about the wrist, especially if the tendons are divided in some place where the tendon sheaths are not sharply defined, the newly formed tissue between the ends of adjacent tendons may unite into one common mass, thus leading to very imperfect function. In the same way, even when only one tendon is divided, it may adhere to the connective-tissue wall of its sheath. In the case of tendons lying in sharply defined sheaths, a large amount of separation is possible. In cases where several tendons lie close together, less separation is possible. In such cases it often is better to fill the gap by some one of the many methods of splicing the tendon, in order to be sure that the line of the tendon is maintained by peritendineum from which the new tendon is to be formed.

E. Repair of Fractures.

The bones, like the tendons, are essentially a modified connective tissue with special functions, the first of which is to furnish support; the second is connected with the function of production of the blood corpuscles. The supporting part of the bones is a modified connective tissue, in which lime salts are deposited. To understand the process of repair of injuries to bone, it is necessary to bear in mind the minute anatomy of the bones.

The bones consist of a supporting framework, rigid from the presence of lime salts, and of a soft central portion, the marrow. On the external surface of the bone is a thin layer of peculiar structure, corresponding to the "bast" of a tree—the "periosteum." This external layer is one of the two actively growing portions of the bone. Lining the inner surface of the supporting portion of the bone are cells which have the same function of bone production as the deeper cells of the periosteum, the "endosteum." The calcified portion of the bone forms an external dense shell, or "cortex," surrounding the more or less open central "marrow cavity." From the inner surface of the cortex irregular beams or "trabeculæ" of bone extend inward, especially near the articular ends of the marrow, forming an irregular meshwork of rigid beams, which adds to the strength of the bone and serves as a support. The trabeculæ make an irregular meshwork of bone, but the spaces of the mesh are not closed spaces, but irregular spaces connecting one with another, although the course of the communication may be very devious. The relative amount of supporting trabeculæ varies in different bones and in different portions of the same bone. In the flat bones of the skull, and in the short bones, the meshwork of the trabeculæ is relatively large and is fairly uniformly distributed. In the long bones, in the articulating end, the trabeculæ are numerous, and form a rather dense internal meshwork of bone, which adds to the strength of the structure. The trabeculæ in this portion of the bone are not arranged irregularly, but are distributed in such a way as to give the maximum of strength, being arranged roughly along the lines of "stress and strain," much as an engineer would arrange the structure of a bridge or derrick. The character of the marrow varies at different ages, and in different bones at the same age. In infants and young people the marrow is red and contains many hæmopoietic cells. In adults the marrow of the long bones is yellow, and consists chiefly of fat cells. The marrow of the short bones more closely resembles the red marrow of children. In old people the marrow often is of a myxomatous structure. The marrow consists of a framework of connective tissue, supporting blood-making or fat cells. The periosteum is a membrane surrounding the cortex, composed of a deep layer of polygonal cells, which have the power of depositing bone, while the outer layer is fibrous, much like dense fibrous tissue. The periosteum is the seat of the peripheral growth of bone. The inner surface of the cortex and the trabeculæ are lined with a membrane of cells (the

endosteum), which have the same function for the internal surface of the supporting framework that the periosteum has for the periphery. Under normal conditions the endosteum may be undemonstrable in ordinary sections, but in bone that is undergoing repair this internal osteogenetic layer is clearly visible. The cortical bone has a laminated structure, and at intervals between the laminæ are open spaces, or "lacunæ," in which lie living bone cells, or "bone corpuscles." The lacunæ communicate one with another by delicate canals, or "canaliculæ," in which run processes of the bone corpuscles. In places in the cortical bone are open canals of larger size, surrounded by concentric bone laminæ, "Haversian canals," in which blood-vessels and nerves run. The spaces between the trabeculæ are the "alveolar spaces."

The bones develop in different ways. Most of the bones are preformed in cartilage, arising from mesoblastic cells, and finally become converted into true bone. This process of ossification in the long bones begins at the middle of the shaft, and extends in both directions toward the ends of the bone. Consequently the ends of the bone persist as cartilage long after the shaft is ossified. These cartilaginous ends form the "epiphyses." The intermediate line between the epiphysis and the ossified shaft, or diaphysis, is the so-called epiphyseal line, and is the point at which new bone is formed to increase the length of the bone.

Some of the bones, notably the bones of the vault of the cranium, are not preformed in cartilage, but are formed directly from mesoblastic cells, without the intervention of cartilage. These are the so-called "membranous bones."

The process of repair by the osteogenetic tissues, although essentially the same as that which takes place after a fracture of bone or after its partial or complete removal, differs from it, nevertheless, in certain details; hence the two processes will be described separately.

Fractures. If a bone is fractured, usually the broken ends are more or less displaced, fragments of bone may lie loose in the tissues, and there usually is more or less stripping of the periosteum, and crushing of the adjacent soft parts, with some hemorrhage; *i.e.*, there is not only an injury of the bone, but also a more or less extensive injury of the soft parts. After a few days a fusiform mass (callus) is formed about the broken ends, and persists for a variable length of time, constantly becoming more rigid and dense. After the ends of the bone are firmly united, the callus disappears more or less completely, and, if the broken ends have been accurately approximated, the external appearance of the bone becomes normal.

The details of the reparative process are best studied in bones in which a loss of tissue has been produced without dislocation or displacement of fragments, *e.g.*, by drilling a small hole vertically into the shaft of a bone of an animal. In this way the process can be studied in its simplest form and the more complicated process of repair in complete fractures will then be more easily understood.

In experimental drill holes the first result of the injury is hemorrhage into

the hole, followed in a few hours by an inflammatory exudate of leucocytes, serum, and fibrin. By the second day a proliferation of periosteal cells and of the endothelium of adjacent blood-vessels begins, both on the outside of, and within, the cortex of the bone. The proliferation of cells external to the cortex arises from the cells of the periosteum at the periphery of the drill holes, and results in the formation of a mass of tissue, thickest over the hole and thinnest at the edges. Among these proliferated periosteal cells are numerous young blood-vessels. This mass of new cells and vessels forms the earliest stage of the "external callus." The internal proliferation arises from the layer of cells of the marrow which lies next to the cortex and trabeculæ (endosteum); it forms a mass of cells about the drill hole, and is thickest opposite the hole. This is the so-called "internal" or "myelogenous callus." In this callus also may be numerous new blood-vessels. Besides the proliferation of osteogenetic cells (periosteal cells externally and the endosteal internally), there is a proliferation of ordinary connective tissue. The cells derived from this ordinary connective tissue cannot at first be distinguished by their appearance from the cells of osteogenetic origin. (See Plate A.)

By the fourth day, in both the external and the internal callus, there appears, between the osteogenetic cells, a homogeneous intercellular substance. This homogeneous substance ("ostoid") marks the beginning of new-formed trabeculæ, for in a short time lime salts are deposited in this material. As the process continues, some of the proliferated cells are retained in the mass of calcified material and become bone corpuscles. Other cells, at the periphery of the calcified, homogeneous material, deposit successive layers of osteoid tissue upon the external surface of the young trabeculæ, producing a steady increase in size. These bone-depositing cells are the osteoblasts. The spaces between the newly formed trabeculæ (marrow spaces) are filled with spindle-shaped young connective-tissue cells, probably not of osteogenetic origin. At this stage the old cortical bone takes practically no part in the process of proliferation, but the drill hole is filled with granulation tissue derived from the periosteum and endosteum.

By the end of a week the exudate usually has almost entirely disappeared. Numerous well-developed young trabeculæ are formed in the external and internal callus. The drill hole is filled with granulation tissue of osteogenetic origin, one part of it arising from the external callus and growing inward, while the other arises from the internal callus and grows outward. In this granulation tissue osteoid tissue appears between the cells and forms the basis of new trabeculæ, which are to replace the defect in the cortex caused by the drill. The cortical bone itself remains practically inert and does not assist in the formation of new bone. The trabeculæ at this period are arranged, in a general way, at right angles to the course of the laminæ of the cortical bone. The surface of the trabeculæ is studded with osteoblasts. Other cells, larger than the osteoblasts, with many nuclei, are fairly numerous at this time. These giant cells or osteoclasts usually lie

in little bays or depressions on the surface of the new trabeculæ, and have the power of dissolving or destroying the bony tissue of the new trabeculæ. As a result of the activity of these two varieties of cells—osteoblasts and osteoclasts—two processes are going on at the same time in the bony portion of the callus: bone formation by osteoblasts, and bone destruction by osteoclasts. Consequently, the form of any given trabeculæ is constantly changing. The tendency of the two processes is so to arrange the new bone that it shall take up weight to the best advantage with the smallest amount of bone. Finally, in the course of three or four weeks, the new trabeculæ which are formed in the granulation tissue between the broken cortical ends become attached to the cortex, and completely fill the defect caused by the drill hole. The external callus, therefore, no longer is necessary to maintain the strength of the bone, and by the action of the osteoclasts is absorbed and nearly or entirely disappears. The same is true of the internal callus. The trabeculæ in the drill hole, by the combined action of osteoblasts and osteoclasts, come to be arranged in the same general direction as the trabeculæ of the original cortex, and repair is practically completed. The bone which fills the drill hole continues to become denser for many months after the injury, and finally it replaces the old bone so perfectly that it is difficult to determine the point of injury. (See Fig. 83.)

In the simplest process of repair of an experimental drill hole, the transition from granulation tissue of osteogenetic origin to bone is direct, without the intervention of cartilage. This process corresponds roughly to the formation of bone as it is seen in the so-called membranous bones. In many cases of simple injury of bone by the drill, however, the process produces granulation tissue of osteogenetic origin, as already described; but this granulation tissue becomes at first converted into cartilage. In this case some of the cells become surrounded by a homogeneous, intercellular material, in which lime salts are not deposited. Some of the cells included in this matrix take on the appearance of cartilage cells, and in this way both external and internal callus may at first be formed of hyaline cartilage to a greater or less extent. Later, as the process advances, this cartilage becomes converted into bone. In fractures in animals, in which the fracture is complete, and probably in all human fractures, this formation of bony callus *via* cartilage is always the course of a greater or less part of the callus.

In complete fractures of bone the general process of repair is the same as that observed in experimental drill holes, but the details vary somewhat. In complete fractures the ends of the bones are nearly always somewhat dislocated, so that perfect approximation of the ends seldom occurs, and as a rule the injury to the soft parts is excessive. When cell proliferation begins it arises internally from the endosteum ("medullary callus"), and externally both from the periosteum and from the connective tissue of the adjacent soft parts ("external callus"). The external callus appears relatively much larger than in drill-hole fractures, and

EXPLANATION OF PLATE A.

Repair of Fractures. (Experimental.) Transverse section through a vertical drill-hole in the femur of a rabbit. Condition after the lapse of eight days. 1, Drill-hole filled with loose fragments of bone and granulation tissue; 2, remnant of fibrin from inflammatory exudate; 3, margin of drill-hole in cortical bone—no proliferation of this dense bone; 4, internal callus, arising from endosteum; 5, 5, external callus, arising from periosteum, and being partly preformed cartilage and partly a direct bony formation; 6, scar tissue in marrow canal, coming chiefly from reticulum of marrow; 7, 7, cortex of femur. (*Original.*)

PROCESSES OF REPAIR

forms a large, fusiform mass, including the fractured ends. At first no distinction can be made between the cells which arise from the periosteum and those which arise from the soft tissues. When trabeculæ form in the callus they may form directly from the osteogenetic granulation tissue, as in the simplest form of drill-hole fracture; or, more often, the first step is a conversion of the granulation tissue into hyaline cartilage. As the process advances, the deeper layers of this cartilage—those nearest the cortical bone—are converted (metaplasia) into bone, and the process continues until the greater portion of the callus be-

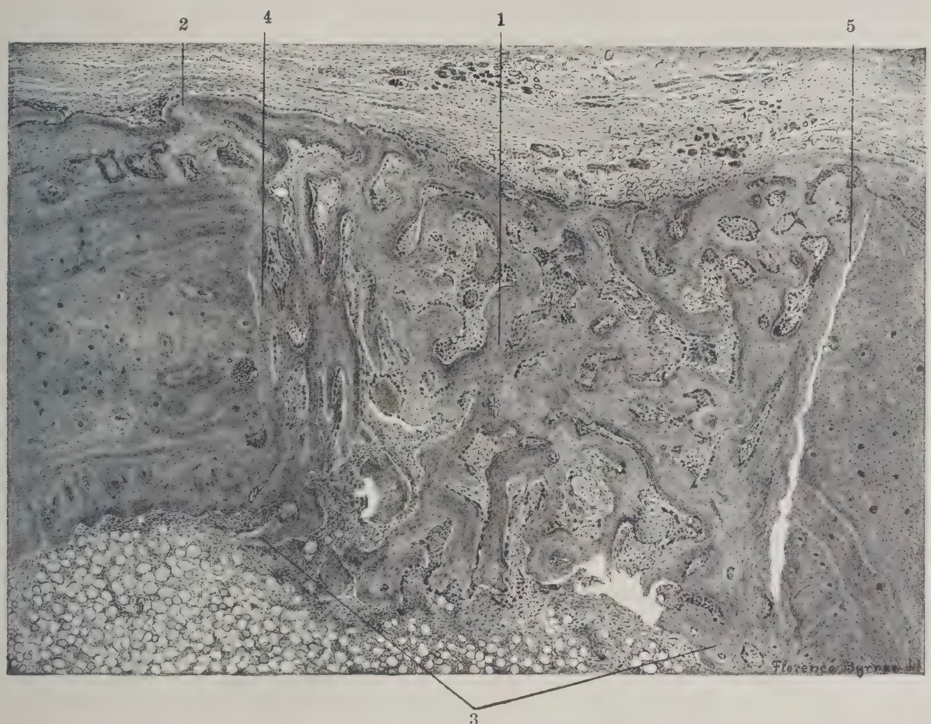

FIG. 83.—Repair of Fractures. (Experimental.) Transverse section through a vertical drill-hole in the femur of a rabbit. Condition after the lapse of twenty days. 1, Drill-hole filled with newly formed trabeculæ, derived from external and internal callus; 2, external callus which has undergone partial absorption; 3, internal callus partly absorbed; 4, new bone which replaces lost bone, adherent to original cortical bone; 5, similar condition, artificially separated from original cortex, showing that union is insecure for some time. Notice that new trabeculæ are beginning to assume a position like that of the original bone. (*Original.*)

comes bone. The same process may be seen, although usually not so well marked, in the internal callus. As in experimental fractures, the cortical bone of the fractured ends remains practically inert, and takes no part in the production of the new trabeculæ, which arise almost entirely from the periosteum and endosteum. The trabeculæ from these two bone-forming layers extend between the broken ends, and finally become attached to the cortical bone, and more or less completely restore the line of the cortex. At first, this new bone is composed of young trabeculæ, studded with osteoblasts and osteoclasts, with narrow spaces between the trabeculæ filled with spindle-celled connective

tissue. The trabeculæ often are arranged at right angles to the line of the laminæ of the cortical bone. Ultimately, the narrow spaces disappear as the trabeculæ increase in size, and by growth of the trabeculæ new dense cortical bone is formed to replace the defect caused by fracture. By the combined action of osteoblasts and osteoclasts, the laminæ of this new dense cortex resume the same general direction as the laminæ of the injured cortex, provided the fractured ends have been accurately approximated.

The ultimate fate of the bone of the external callus depends upon the accuracy of approximation of the fractured ends of the bone. If they are so approximated as practically to restore the original contour of the bone, after a time there is marked or complete absorption of the external callus, and the bone resumes its original contour. If, however, the dislocation of the fractured ends is extreme and is not reduced, much of the external callus persists after bony union has taken place, and the laminæ of the callus which persists are not parallel to the laminæ of the unbroken cortex, but are arranged in such a way as to take up weight to the best mechanical advantage. A persistence of a marked amount of external callus always indicates malposition of the fractured ends. This fact is of great clinical importance. The time required to repair any given fracture depends upon the size of the fractured bone, upon the accuracy with which the ends are approximated, and upon the care and perfection with which they are immobilized.

In some cases bony union is delayed for long periods, or, indeed, may never take place. In many cases the reason for failure to unite is not clear. Sometimes it appears to be due to the inclusion of soft tissues between the broken ends, thus preventing the union of the two sides of the external callus. This cause, however, certainly is a rare one. In other cases tissue having the structure of bone (osteoid tissue) forms an external callus, but no deposit of lime salts takes place. The cause of this is unknown. In some cases the fractured ends are united by dense scar tissue only, without any bone formation. This produces a flail-like joint, or "syndesmosis." Or one fractured end may enlarge and form a false socket, while the other end forms a false head, both contained in a capsule of dense fibrous tissue, forming a sort of synovial cavity, or false joint ("pseudo-arthrosis"). The two articulating ends generally are covered with a layer of dense fibrous tissue, and not with cartilage. Sometimes, when two adjacent bones are broken, the two calluses may unite to form one single callus, and thus the two bones are firmly united ("synostosis").

The gross appearance about a fracture corresponds to the histological condition already described. The swelling of the soft parts which appears at the end of a few hours is due to the presence of an inflammatory exudate in the injured soft tissues. The blebs and bullæ which may appear in a short time are due to elevation of the superficial layers of the skin by the fluid exudate, which extends toward the surface. If the injury to the soft tissues is severe, the overlying skin

may be discolored bluish at once from deep hæmatoma, or it may become black and blue after a few days from disintegration of a deep hemorrhage, with diffusion of blood pigment. As the exudate diminishes the tissues about the broken ends become thickened from cell proliferation, and form a spindle-shaped thickening (granulation-tissue external callus), composed of ordinary granulation tissue and of granulation tissue arising from the periosteum. At first, this callus is firm and elastic, but not bony. After about two weeks the callus obviously becomes harder and more sharply defined (ossification of deep portion of callus derived from periosteum). At this time the fragments, which at first are freely movable, are much less so, and move only under strong pressure. After a variable number of weeks, depending upon the site and severity of the fracture, mobility entirely disappears (restoration of cortical defect), although the callus persists. At this time the bone is strong enough to bear weight. In the course of months the callus progressively becomes smaller, and finally may largely or entirely disappear if the bones are in perfect position. The amount of callus which persists is proportional to the amount of deformity.

There are certain principles of treatment of fractures which depend upon the above-described conditions.

The first essential is to secure as perfect as possible approximation of the fractured ends. The more perfect the position of the fracture, the smaller the external callus and the shorter the time required for completion of repair of the bone. The more perfect the position, the less is the interference with the soft tissues, and in all fractures it is to be remembered that there is injury not only of bone, but of soft tissues. As regards the reduction of the deformity, it should be borne in mind that attempts at reduction more than two weeks after injury are likely to give poor results. During the earlier stages the callus is soft and not ossified. After the second week bone formation is well advanced, the ends of the bone are included in the spindle-shaped callus, are not freely movable as at first, and forcible correction causes injury to the newly formed bone and prolongation of the process of repair.

Perfect immobilization also is essential. The less the callus is interfered with, the greater is the rapidity of repair. In case of fracture about tendons or into the articular surfaces of joints, other mechanical problems enter in, so that early mobility may be necessary and the importance of rapid ossification may hold a secondary place.

Regeneration of bone. As has been said already, growth in diameter of bone is dependent upon the periosteum. The calcified bone itself is practically inert. In cases in which there has been extensive destruction of bone, advantage may be taken of the power of the periosteum to produce new bone to replace loss.

Ollier has shown that if the entire shaft of a healthy bone of an animal be removed subperiosteally, leaving the periosteum intact, the periosteum will produce new bone exactly similar in outline to that portion of the bone which

has been removed. In the same way, if an entire diaphysis, *e.g.*, of a long bone, is destroyed by disease, *e.g.*, by acute suppurative infection (acute suppurative osteomyelitis), advantage can be taken of this fact to bring about a complete regeneration of bone to replace the lost tissue.

The vitality of calcified bone is very much lower than that of the surrounding periosteum. Various diseases (osteomyelitis, tuberculosis, sarcoma) may cause the death of a considerable portion of any bone. The dead bone loses its power of performing its function of supporting weight. In that case the periosteum surrounding the necrotic bone proliferates to form new bone to take up the weight-carrying function. The dead bone persists as a foreign body, while the periosteum forms a cylindrical layer of new bone, of a structure like that of the bone seen in the external callus, about the dead bone. The dead bone persists as a "sequestrum," surrounded by a cylindrical "involucrum" of new periosteal bone. The involucrum at first is soft, like the early external callus, and continues to thicken until the diameter of the new bone equals or somewhat exceeds the diameter of the original shaft. As the involucrum becomes older it becomes denser, like ordinary cortical bone, which, as has already been shown, has very limited power of repair. The sequestrum usually is connected with the surface of the body by various "sinuses," which perforate the involucrum at various points. At any of these stages, *i.e.*, early necrosis, early periosteal proliferation, or in the stage of involucrum and sequestrum, it is possible to take advantage of the regenerative power of the periosteum and endosteum to bring about complete regeneration of bone.

In all cases it first is necessary to remove the necrotic bone, which acts as a foreign body. After the necrotic bone has been removed, the intact periosteum should be approximated so as to bring the internal surfaces together and to leave no central cavity. The growth of the periosteum is peripheral, and new bone, like the external callus, is formed, until there is produced a shaft of periosteal bone which slightly exceeds in size that of the original shaft. As the bone becomes harder as it grows older, there is some absorption of the bone, until ultimately the new bone is of the same size as the original shaft. The new bone at first is solid bone without a marrow canal, but finally, so far as can be judged from *x*-ray pictures, there is an absorption of the bone in the centre of the shaft, and a new marrow canal is formed. The notable thing about this process of bone regeneration by the periosteum is that the new bone is of exactly the same shape as the original bone, and cannot be distinguished from it even by touch, sight, or the *x*-ray. This suggests that the shape of the bones of the human skeleton is due to two causes—heredity and environment, or function. Hence when a bone is removed the new bone which is formed is of the shape which performs function to the best advantage. This is true of very complicated bones, and even of complicated joints which are excised subperiosteally.

In some cases in which the involucrum is old it has limited power of repair,

and in such cases both involucrum and sequestrum must be removed, to give the periosteum a chance to form an entirely new bone.

F. Repair of Muscle.

After a wound of striated muscle there comes, as in all injuries of the soft tissue, an inflammatory exudate. In the course of a few hours there arises a new growth of granulation tissue from the adjacent connective tissue and also a peculiar series of changes in the muscle itself. Some of the muscle fibres next to the wound become necrotic; they are invaded by polynuclear leucocytes and endothelial cells, dissolved, and removed. In some of the other muscle fibres there occurs an increase in the number of the nuclei, which arise not by mitosis, but by direct nuclear division. These nuclei arrange themselves in the ends of the muscle fibres, and, instead of having a mural arrangement like that of the nuclei in normal muscle fibres, are situated in the middle of the fibre. The fibre itself loses its striæ, and becomes more or less fibrillated longitudinally. The greater portion of these cells finally disappear, so that in the granulation-tissue scar only an occasional club-ended fibre is left, and the defect in the muscle is replaced by granulation tissue, which ultimately becomes scar tissue. If the ends of the muscle are accurately approximated, the scar is a small one and interference with muscle function is slight. If the ends of the muscle are widely separated, there may be great impairment of function.

G. Repair of the Heart.

In wounds of the heart the muscle fibres take no part in the process of repair, but the defect is filled by granulation tissue, which finally forms a scar. Adhesion to the pericardial walls is common.

H. Repair of Blood-Vessels.

Wounds of vessels of large size present a condition somewhat different from that of wounds of other tissues, since the walls contain no small vessels except in the adventitia coat, so that the early adhesion of the edges of the wound is not produced by an inflammatory exudate in the ordinary sense of the word, but is due to fibrin which arises from the circulating blood in the vessel itself. The conditions vary somewhat in arteries and veins, and with the character of the injury, *i.e.*, whether there is complete division of the vessel, or a lateral wound, or a rupture of the internal coat.

In arteries complete division of the wall by a sharp instrument of course leads to violent hemorrhage, which may cause death in a short time. In complete division of an artery by tearing or by similar violence, however, extensive hemorrhage as a rule does not take place, and may be absent even in clean cuts, because the ends of the vessel retract into the surrounding tissues, while the walls of the vessel become occluded by the formation of a clot, composed of fibrin derived from the blood in the vessel and enclosing red blood globules and a few leuco-

cytes. In wounds in the wall of an artery the hemorrhage takes place into the soft tissues about the point of injury, and coagulates, so that finally the edges of the vessel wound are sealed together by a layer of fibrin. Within the lumen of the vessel there may be simply a thin peripheral clot at the point of injury, covering the wound; or in other cases, especially if the endothelium is extensively injured, a thrombus may form, of such size as to occlude the vessel. In ligature of a vessel which is completely divided there is formed, at the point of ligature, a clot, the size of which is variable, depending upon the rapidity of circulation, the amount of injury to the endothelium, and the perfection of the asepsis.

After the formation of the thrombus the later stages are like those of any wound; *i.e.*, the clot, which is essentially an inflammatory exudate, in which red blood globules are overwhelmingly predominant, becomes converted into organized tissue. If the clot is a small one, situated peripherally, the surface may be covered by newly formed endothelial cells, while the deeper layers of the clot are replaced by newly formed connective tissue derived from the media and adventitia. If the clot is of large size and completely fills the lumen of the vessel, the surface of the clot toward the blood stream is covered with endothelium growing from the walls of the vessel, while the clot itself becomes organized by granulation tissue. The lumen of the vessel beyond the point of obstruction undergoes a slow diminution in size through an obliterative endarteritis.

In some cases, in which the wound in the vessel is a lateral one, a large clot forms about the point of injury and pushes the surrounding soft tissues to one side, until the pressure becomes so great that no further hemorrhage takes place. The effused blood coagulates, and finally the periphery may become organized by granulation tissue arising from adjacent connective tissue. In some cases the centre of this area may remain patent and contain fluid blood, connected with the circulating blood in the patent vessel through the interval in the wall made by the wound, thus forming a false traumatic aneurism.

In complete division of veins the divided ends usually are filled with a blood clot which becomes covered by endothelium, while the clot itself comes to be replaced by dense scar tissue derived from the media and adventitia. In some cases, however, the amount of terminal clot is exceedingly small. In case of a lateral wound of a large vein, it often is possible to prevent severe hemorrhage by ligaturing the wound in the vessel. In that case the inner wall of the veins is puckered by ligature, and may be covered with a thin, peripherally placed clot, which may become organized without the formation of an obstructing and obliterating thrombus.

In all lateral wounds of vessels absolutely perfect asepsis is essential if one expects to obtain healing without complete thrombosis and obstruction. The presence of even a slight amount of infection is practically certain to cause sufficient injury to the endothelium to produce complete obstruction. In suturing

of vessels it is said that the projection of perfectly aseptic sutures into the lumen of the vessel through the endothelium does not necessarily produce thrombosis, but that in many cases the sutures are very early covered with new endothelium.

I. Peripheral Nerves.

Section or destructive injury of a peripheral nerve causes an immediate traumatic local degeneration of the nerve at the point of injury. This is followed by a degeneration throughout the extent of the nerve peripheral to the point of injury, and a degeneration of the fibres proximal to the point of injury, extending no farther than the first few nodes of Ranvier. There also occur changes in the cells of origin of the degenerated nerves, resulting in an effacement of the granular structure of the nerve cell body, with displacement of the cell nucleus to the periphery of the cell—the so-called "axonal reaction" of Nissl.

Following the degeneration occur regenerative changes in the nerve, which may lead to a restoration of function. The extent to which this regeneration may occur depends somewhat upon the amount of injury to surrounding soft parts. If the injury is one which destroys the integrity of the nerve fibre, without destroying the continuity of the nerve sheath—*e.g.*, crushing injuries—the regeneration of the nerve is more rapid and certain. If the nerve is cut across and the ends are sutured together, regeneration is more likely to occur than it is if the ends retract and become widely separated, or if suppuration occurs so that the ends are separated by a wide zone of granulation tissue. If the peripheral end of a nerve is entirely removed, as, *e.g.*, in an amputation, a peculiar partial regeneration of the proximal portion may occur, resulting in an "amputation neuroma."

After the receipt of an injury there comes a traumatic degeneration of the nerve in the immediate vicinity of the injury. The amount of this degeneration depends upon the character of the injury, being, *e.g.*, slight in a clean-cut wound and more extensive after a crush. Immediately after this change there comes a secondary ("paralytic") degeneration of the nerve, extending in either direction from the point of injury. On the central side of the injury the degeneration extends upward to the nearest nodes of Ranvier. On the peripheral side the degeneration extends throughout the entire extent of the nerve.

The degenerative changes produce a fragmentation and fibrillation of the axis cylinder, and a fragmentation of the medullary sheath. Very early there also arises marked proliferation of the cells in the sheath of Schwann.

The regenerative process begins after the degenerative process. It is difficult to say just how the new axis cylinders are produced, there being dispute upon this point; but the new axis cylinders extend gradually into the peripheral end. Most observers believe that the process of growth is like that in embryonal development, *i.e.*, there is a constant peripheral growth. Others believe that the

new formation is, partly at least, the result of activity of cells in the sheath of Schwann. As a practical matter, the new fibres in the adult always arise from the central stump and extend peripherally along the track of the original nerve.

The new fibres, as they arise from the central stump, tend to split into bundles of small neuro-fibrils, of which the original nerve is supposed to be composed. The direction of the new fibres may be modified by various mechanical obstructions, and also by an apparent attraction of the distal nerve remnant for the proximal nerve fibres. The fibres at first grow in the interstices between the cells of the scar ("neurotization of the scar"), which lies between ends of the nerve. If the scar between the ends is too dense, the new fibres may grow into the tissues in various directions, and never may be able to get into contact with the peripheral stump. In such cases no restoration of nerve function takes place. The tendency of the proximal axones to join the peripheral stump can be favored by various mechanical means, *e.g.*, by the introduction of catgut sutures or hollow tubes, along the tract of the nerve, or by means of neuroplastic flaps. Regeneration is obstructed by secondary infection with excessive formation of granulation tissue.

The rate of regeneration varies somewhat, but is approximately at the rate of 1 mm. per day.

J. Central Nervous System.

As regards regeneration or repair of injuries to the central nervous system, while theoretically possible to a very slight degree, the amount of regeneration is so slight as to be of no surgical importance. The cause of the lack of power of central nerves to regenerate is obscure, but it is claimed to be due to the fact that the central nerve fibres do not possess a sheath of Schwann, which is essential in some way to the new formation of axis cylinders, and also to the fact that the neuroglia fibrils offer a mechanical obstruction to the advance of nerve fibres.

TUMORS AND TUMOR FORMATION.

By ALBERT G. NICHOLLS, M.D., C.M., Montreal, Canada.

Definition.—The term *tumor* in its literal sense means *swelling*. Any swelling, therefore, irrespective of its cause, might be called a tumor. Swelling, however, as we know, is merely an external symptom and may be brought about by a great variety of causes, such as congestion, œdema, hemorrhage, inflammatory infiltration, deposits of various kinds, and the new formation of tissue. All the conditions mentioned have this in common, that the part is enlarged. In the old days, before the publication of "Die krankhaften Geschwülste," the wildest speculations were rife as to the causes of pathological phenomena, so that we are not surprised that many essentially unlike conditions should have been confused together. The word "tumor" was conveniently broad and noncommittal and, like charity, was made to cover a multitude of sins. It is curious how traditional modes of expression will persist, for even yet we not infrequently speak of *tumor albus*, the *white swelling* or *tumor*, when we mean tuberculous synovitis with effusion. The appearance, in 1863, of Virchow's epoch-making work, with its insistence on the doctrine of what is commonly known as the "cellular pathology," laid the foundation of and pointed the way to a more adequate conception of pathological processes, particularly cell proliferation. From this time modern pathology may be said to date. With the improvements in microscopical technique many additional facts have been recorded, and while many of Virchow's conclusions have been shown to be partial and even erroneous, the fundamental principles which he laid down have been confirmed and strengthened. The result has been to restrict the term *tumor* to pathological new-formations of tissue. But here a difficulty was soon encountered, a difficulty that cannot be said to be entirely cleared up even yet. This is, that certain inflammatory processes give rise to local swellings and some of the other phenomena that we usually associate with the idea of a tumor. Thus, in typhoid, malaria, and some other infectious diseases the spleen may be greatly enlarged as a result of proliferation of tissue. The most notable example is, however, to be found in the so-called "infective granulomata." In tuberculosis, syphilis, actinomycosis, leprosy, and some forms of animal parasitism, we get localized nodules, associated often with great proliferation of cells, with central necrosis, which tend to spread and may even give rise to similar growths elsewhere. The resemblance to a tumor is therefore striking. More thorough investigation has served to draw a distinction between cell proliferation, due to infective and other forms of irritative

inflammation, and tissue neoplasia due to none of these causes. What, then, constitutes the difference between an inflammatory neoplasm or granuloma and a tumor, using the latter term in its more restricted modern sense? An inflammatory granuloma can be traced to a definite cause, usually some micro-organism; it is reactive and its purpose benign, in so far as it is an attempt to neutralize the effect and repair the damage caused by the invading element; the process goes on only so long as the cause is operative, and ceases when it has come to an end. A true tumor, on the other hand, is a new formation of tissue, due to no demonstrable cause; the vegetative power of the cells composing it is excessive and appears to be inherent; the growth takes place without regard to the neighboring structures and is, therefore, a law unto itself; finally, it subserves no useful purpose in the body. We may, therefore, with Thoma, define a tumor shortly as *an autonomous* or *independent new-growth.* The peculiar features of tumors are the following: (1) The majority begin at some one point in an organ and subsequently spread to neighboring parts. (2) They reproduce with more or less modification the tissues from which they spring. (3) They differ in physiological function from the part in which they are found. (4) They cause pressure-atrophy and dislocation of the adjacent structures, or, again, lead to destructive infiltration. (5) They are particularly liable to retrogressive changes. (6) The tumor cells in many instances, when transplanted to distant parts, give rise to secondary tumors resembling in properties and appearance the original growth.

Etiology.—Notwithstanding the fact that of late years our information in regard to tumors and tumor formation has been steadily increasing, the question of etiology still remains largely an unsolved problem. We know to some extent the general laws governing the proliferation of tissue. We are familiar with the appearance and minuter structure of the various tumors. We can apprehend in some degree their mode of origin and method of extension. We have made some progress in differentiating the various forms. But, when all is said and done, it must be confessed that the essential cause has up to the present eluded discovery. We are still puzzling over the question, What is the force that in the first instance determines the cell proliferation in tumors? I do not propose here to enter the arena of controversy and discuss the various theories that have been advanced to explain tumor formation. This has been done very fully and competently in another portion of this work. I will, therefore, content myself simply with drawing attention to a few points that are of considerable practical importance.

Leaving for the moment the benign tumors out of consideration, the surgeon is confronted by two undeniable facts. Carcinomata develop most frequently at the so-called ostia of the various portions of the alimentary tract, the lips, tongue, cardia and pylorus of the stomach, the ileo-cæcal valve and anus; in ducts and hollow viscera, as, for instance, the bile ducts, the gall bladder, the urinary bladder,

and the uterus. Tissues in all these places are subject to considerable mechanical and other irritation. We may conclude, therefore, that the influence of external traumatism, using that term in its widest sense to include irritation from mechanical, thermal, chemical, and infective causes, is by no means unimportant. A great deal of evidence has accumulated to support this position. Carcinomata have, for instance, been known to develop in the cicatrices of burns, in the neighborhood of setons, in the bases of chronic ulcers and lupus patches, and at the orifices of sinuses. The irritation of soot (sweep's cancer), tar, and paraffin in the clothing occasionally sets up carcinoma of the scrotum. Epitheliomata of the lip and tongue are not infrequently associated with irritation of the part by a pipe; carcinoma of the biliary passages is often accompanied by cholelithiasis; a chronic ulcer of the stomach may become malignant. Again, constant or repeated irritation may convert a benign growth into a malignant one. Yet, when we consider how often irritation of the same kind and intensity fails to produce tumor growth, we have to admit that irritation can only be the exciting cause, and that at the back of it all is some unknown force that determines the fact of cell proliferation.

The second point is, that many tumors arise in parts of the body where there are transition of epithelium, complicated infoldings of tissues, and the closure of developmental fissures. At such places the cell equilibrium appears to be unstable. As examples may be cited: cystic tumors and epitheliomata occurring in the neck in parts where normally epithelium does not exist, in consequence of defective closure of the branchial clefts; hypernephromata; the heterologous tumors, such as chondromata of the mamma, parotid, and testis; dermoid cysts of the ovary and testis, and other teratoid growths. In many cases it can be shown that the neoplasm originates in misplaced embryonic cells or "rests." With the increase in our knowledge of tumors, this class of growths has been greatly enlarged, and the "developmental" theory of tumor formation has probably the greatest number of adherents among pathologists. None, however, give it the wide application that Cohnheim has done. Not a few tumors have not as yet been satisfactorily accounted for on this basis, and in any case, even were the theory universal, the ultimate cause of the neoplasia remains unknown.

The Gross Appearance of Tumors.—Tumors vary greatly in size. Some are microscopic, others may exceed in weight the individual in whom they are found. The shape is also variable, being in large part governed by external conditions. Tumors on free surfaces grow in all directions and tend to assume a rounded form. Those occurring in closed cavities accommodate themselves to the space in which they lie. Tumors may, therefore, be tuberous or nodular, lobulated, fungoid, polypoid, papillary, sessile, or diffuse.

In regard to consistence, some are soft, friable, juicy, and brainlike; others firm, hard, fibrous, or stony. Differences in consistence and texture may be found in different parts of the same tumor.

Most new growths are white or grayish-white in color. Some, however, are reddish, yellow, brown, green, or even black. As a rule, the substance of a tumor is sufficiently unlike that of the part in which it is found to render its detection easy. Yet some, like the gliomata of the central nervous system, are with difficulty distinguished from the healthy tissues.

The benign neoplasms are provided with a more or less complete capsule, while the malignant ones are badly defined and infiltrating in character.

On section, tumors may present the features above mentioned, but may also in parts show evidences of retrogressive processes, fatty degeneration, caseation, liquefaction, hemorrhagic extravasation, colloid transformation, or even suppuration.

The Classification of Tumors.—There are few subjects in the realm of pathology more fraught with difficulty than this. For, as in so many other branches of science, so here, improved methods of investigation and more extensive information have resulted in the replacement of the crude ideas formerly in vogue by much more complex conceptions. Yet we cannot say that the steadily increasing knowledge we are gaining from day to day with regard to the structure and histogenetic development of new growths has been attended by a corresponding advance in our views as to the true nature of these formations. Various classifications, as numerous as the definitions of what constitutes a tumor, have been proposed, all of which have, in the light of modern investigations, been proved to be faulty in one or more important particulars. Four methods of classification appear to be possible: (1) According to *etiology*, (2) according to *clinical peculiarities*, (3) according to *morphology* and *histogenetic development*, and (4) on the basis of *embryological differentiation*.

A classification on etiological principles, were it possible, would be strictly scientific, but it is hardly necessary to say that, in view of the obscurity that enwraps the question of the essential cause of neoplastic growth, such a classification is not at present practicable, nor, indeed, in my opinion, is it likely to be so useful as some others that might be devised. We may, then, dismiss this part of the subject without more ado.

The clinical behavior of tumors enables us to lay down certain broad generalizations that are unquestionably of value and convenience in any consideration of the nature of neoplastic growth. I refer to the common division of neoplasms into *benign* and *malignant*. A benign tumor may in general terms be defined as a slowly growing tumor, often encapsulated, which does not tend to invade neighboring structures, does not produce secondary growths in distant parts, and produces its symptoms chiefly by its bulk. A malignant tumor, on the other hand, is usually rapid in its growth, invades and destroys the adjacent tissues, is apt to form secondary growths in other organs, tends to necrose or ulcerate, frequently recurs after removal, and, finally, produces certain grave constitutional disturbances commonly included under the term *cachexia*. This class-

ification, while to a certain extent it subserves a useful clinical purpose, is open to the objection that it lays stress on a somewhat inconstant feature of tumors as a means of differentiation, to the exclusion of much more fundamental characters. As a consequence, tumors that have little in common in point of structure are brought into the same category. We know, for example, that chondromata and adenomata, both of which are usually regarded as benign growths, may, exceptionally, give rise to secondary growths, which, however, do not markedly tend to infiltrate. In other words, they occasionally exhibit a limited tendency to malignancy. Again, certain tumors, notably pigmented moles, may be practically identical in histological structure with melanotic sarcomata and carcinomata, and yet may persist for years without taking on excessive growth, though they may do so in time, when irritated. Their malignancy is, as it were, latent. Their place, therefore, seems to be intermediate between benign and malignant growths. The classification in question is, in fact, too wide to be accurate and is, moreover, as artificial and unscientific as the Linnæan classification of plants.

Much more can be said in favor of the grouping of tumors on a morphological basis, and this is the method which, in some shape or other, is the most popular among pathologists at the present time.

With the advent of the newer "Cellular Pathologie" it became possible for the first time to attempt the classification of new growths on a rational basis, and with the improvements in technique, and the enormous increase in knowledge that has resulted therefrom, the principles originally enunciated by Virchow have been in a large measure placed upon a solid substratum of fact.

Virchow, among other things, as a result of his investigations, pointed out that certain tumors conform more or less accurately, so far at least as their structure is concerned, with normal forms of tissue, while others show deviations, chiefly in the direction of being more cellular. The latter for the most part are malignant in nature with all the peculiarities which this implies. According to Virchow, then, we can recognize two main groups, the *cellular tumors* and the *less cellular tumors*. In the former, the component cells are greatly increased as compared with the corresponding normal tissues. This includes forms which are now termed sarcoma and carcinoma. The latter group was subdivided by Virchow into the *histioid* and *organoid* tumors. Histioid tumors are made up for the most part of cells resembling one of the normal tissues, while organoid tumors are composed of several tissues, normal in regard to their structure, and arranged after the fashion of an organ. This classification is important chiefly because it clearly indicates the agreement between normal and autonomous tissue formation. More complete study has shown, as Thoma has pointed out, that no tumor consists of only one tissue. All, for example, are provided with blood-vessels, and many of them with nerves, while at some part there is invariably a certain amount of connective tissue to be found. Hence, histioid tumors always have an arrangement similar to that of an organ, if it be only to a limited degree. The

distinction between histioid and organoid tumors may properly. therefore, be allowed to drop.

From somewhat different considerations, Virchow again recognized two great classes of tumors: the *homoplastic* growths, which in structure closely resemble the normal tissues from which they arise, and the *heteroplastic*, which deviate widely from the normal. More complete investigations have shown, however, that perfect homoplasia never really exists, and that all true tumors—and in this category we would not of course place ordinary tissue hypertrophies—are to a greater or less degree heteroplastic.

If it be granted—and for these conclusions we have ample proof—that every cell or group of cells is derived from some pre-existing cell ancestor, and, as a corollary to this, that every new growth has its prototype in a normal tissue, then it is possible to classify tumors according to their origin, and this is the fundamental principle underlying what may be called the *embryological* method of classification.

It is now well recognized that at a very early period in the development of the embryo the almost entirely undifferentiated cells of the morula become arranged into two layers, the primitive *epiblast* and *hypoblast*, indicating the future epiderm and endoderm. Very soon the hypoblast, or innermost of the two primitive layers, proliferates and gives rise to a mass of cells, the *mesoblast*, which lies intermediate between the primitive epiblast and hypoblast. To these three primitive layers, epiblast, mesoblast, and hypoblast, can be traced the origin of all the tissues of the body. This being so, it is quite logical to classify tumors in the same way, and this in fact has been done, notably by Waldeyer, who recognized tumors of epiblastic, mesoblastic, and hypoblastic derivation. It was quickly found, too, that epiblastic and hypoblastic tumors presented many striking points of similarity, so that they have now come to be classed together under the designation of tumors "of epithelial type," as contradistinguished from those of mesoblastic origin and "connective-tissue type." Thus, both on embryological and on histological grounds, we can recognize two great classes of tumors, apparently sharply differentiated the one from the other, those of mesoblastic origin and connective-tissue type, and those of epithelial and glandular origin and of epithelial and glandular type. This is the most popular classification among pathologists at the present time, is withal practical and, so far as it goes, scientific. Both groups may be divided into benign and malignant.

The benign connective-tissue growths include the *fibroma, myxoma, lipoma, myoma, chondroma, osteoma, glioma, neuroma, hæmangioma, lymphangioma.*

The malignant are the various forms of *sarcoma*, and the *malignant myomata.*

The benign tumors of epithelial type include the *papilloma*, the *adenoma*, and *cystadenoma.*

The malignant are the *malignant adenoma*, the *adeno-carcinoma*, *carcinoma*, and *epithelioma.*

To these may be added certain mixed forms, consisting both of epithelial and of connective-tissue elements, one or other of which may predominate. Such are the *papillary fibroma*, the *papillary cystadenoma*, the *adeno-fibroma*, and similar growths.

It may be remarked *en passant* that the majority of epithelial tumors are, in a sense, of mixed type, for, with the possible exception of the epithelioma, most of them show evidences in some part or other of a new formation of fibrous tissue.

The objections that have been brought forward to this mode of classification are that there are not a few cases to be met with which do not fit into the scheme. The first great type embraces the tumors of mesoblastic origin and connective-tissue type. Now, the gliomata, which are of connective-tissue type, and, therefore, are generally classed with the fibromata and other tumors of this group, are not mesoblastic, but epiblastic. To include them with the fibromata and homologous growths is, to say the least, artificial. Again, certain tumors of the kidney, suprarenal, ovaries, testis, and uterus, while histologically of epithelial type, in that resembling the carcinomata, are really of mesoblastic origin. If, therefore, we are to preserve the embryological method of classification, some method must be devised of grouping like with like, and bringing histological structure into harmony with embryological derivation.

This has been attempted by Prof. J. G. Adami in an important contribution to the subject, entitled "On the Classification of Tumors" (*Journal of Pathology and Bacteriology*, June, 1902). He recognized that in early fœtal existence we have two differentiations of the primitive cell layers, leading to the production of two sets of tissues. One he calls *lepidic* or *lining-membrane* tissues; the other, *hylic* or *pulp* tissues. The lepidic tissues form the lining endothelium of blood-vessels, lymphatics, serous membranes, and the acini of various glands. They have this in common, that there is an absence of stroma between the members of the cell groups. The pulp tissues are composed of an intercellular ground substance, either homogeneous or fibrillated, separating the specific cells of the tissue, and constitute the supporting stroma.

On this basis, and in accordance with the principles just enunciated, Adami would classify blastomatous tumors after the following scheme:

I. LEPIDOMATA OR "RIND" TUMORS.

A. Primary Lepidomata.

1. Epilepidomata.

Tumors whose characteristic constituents are overgrowths of tissues, derived directly from the epiblastic lining membranes, or true epiblast.

(*a*) *Typical.*—Papilloma, epidermal adenomata (of sweat, salivary, sebaceous, and mammary glands, etc.).

(*b*) *Atypical.*—Epithelioma proper, carcinoma of glands of epiblastic origin.

2. Hypolepidomata.

(*a*) *Typical.*—Adenoma and papilloma of digestive and respiratory tracts, thyroid, pancreas, liver, bladder, etc.

(*b*) *Atypical.*—Carcinoma developing in the same organs and regions.

B. Secondary Lepidomata.

3. Mesolepidomata.

Tumors whose characteristic constituents are cells derived in direct descent from the persistent *mesothelium* of the embryo.

(*a*) *Typical.*—Adenoma of kidney, testicle, ovary, urogenital ducts; adenoma of uterus and prostate; adenomas originating from the serous membranes, "mesothelioma" of pleuræ, peritoneum, etc.

(*b*) *Atypical.*—Cancer of the above-mentioned organs; squamous endothelioma, so called, of serous surfaces; epithelioma of vagina.

4. Endothelial Lepidomata.

Tumors originating from the endothelium of the blood- and lymph-vessels; endothelioma, perithelioma.

II. HYLOMATA OR "PULP" TUMORS.

1. Epihylomata.

Tumors whose characteristic constituents are overgrowths of tissues derived from the embryonic pulp of epiblastic origin.

(*a*) *Typical.*—True neuroma, glioma.

(*b*) *Atypical.*—"Glio-sarcoma."

2. Hypohylomata.

Tumors derived similarly from embryonic pulp of hypoblastic origin. (?) Chordoma.

3. Mesohylomata.

A. Mesenchymal Hylomata.—Derived from tissues originating from the persistent mesoblastic pulp or mesenchyme.

(*a*) *Typical.*—Fibroma, lipoma, chondroma, osteoma, myxoma, leio-myoma.

(*b*) *Atypical.*—Sarcoma (derived from mesenchymatous tissues), with its various subdivisions, fibro-sarcoma, spindle-cell sarcoma, oat-shape-cell sarcoma, chondro-sarcoma, osteo-sarcoma, myxo-sarcoma, melanotic sarcoma, etc.

B. Mesothelial Hylomata.—Tumors which are overgrowths similarly of tissues derived from embryonic pulp of definitely mesothelial origin.

Rhabdomyoma.

It will, perhaps, be an aid to the proper understanding of a somewhat abstruse part of the subject, and make clear the virtues of the new classification, if we

enumerate the various tissues and structures derived from the primitive germ layers.

I. Epiblastic Structures.

The skin and its appendages, epidermal glands, hair, nails, enamel of the teeth, the lens of the eye, the epithelium of the cornea, olfactory organ, the membranous labyrinth of the ear, the epithelium of the mouth, salivary glands, buccal portion of the hypophysis cerebri; the epithelium of the anus and male urethra, with the exception of the prostatic portion; the central, peripheral, and sympathetic nervous systems; the retina; neuroglia.

II. Hypoblastic Structures.

The notochord; the epithelium of the digestive tract and associated organs, œsophagus, stomach, intestines, liver, pancreas; the specific cells of the tonsils, thymus, and thyroid glands, parathyroids, pharynx, and Eustachian tube; the epithelium of the respiratory tract, larynx, trachea, and lungs, of the bladder, female urethra, and the prostatic portion of the male urethra.

III. Mesoblastic Structures.

1. Mesothelium.

The lining cells of the pleuræ, pericardium, and peritoneum; the specific cells of the suprarenals, kidneys, testes, ovaries (Graafian follicles); the epithelium and glands of the Fallopian tubes, uterus, vagina, vasa deferentia, vesiculæ seminales; striated muscles, including that of the heart.

2. Mesenchyma.

Fibrous connective tissue, cartilage, bone, reticulum of lymph nodes, bone marrow, fat, unstriated muscle, spleen, the endothelium of blood-vessels and lymphatics, blood corpuscles, the endothelium of the arachnoid, synovial, bursal, and corneal spaces; nerve sheaths.

I. Tumors of Epiblastic Origin.

Adenomata and cystadenomata of the epidermal glands and epithelium of the tooth papillæ; epidermoids (cholesteatoma) and inclusion dermoids; epithelioma; neuroma; glioma.

II. Tumors of Mesoblastic Origin.

(*a*) *Mesothelial.*—Adenoma and cystadenoma; carcinoma; rhabdomyoma; hypernephroma.

(*b*) *Mesenchymatous.*—Fibroma, myxoma, lipoma, chondroma, osteoma, leiomyoma, angioma, myeloma, endothelioma (perithelioma) of blood-vessels and lymphatics; sarcomata of all kinds.

III. Tumors of Hypoblastic Origin.

Papilloma, adenoma, chordoma (?), carcinoma.

The accompanying figure (Fig. 84), taken from Adami's paper above referred to, illustrates in a graphic way the differentiation of the various embryonal tissues, at the same time indicating their function and relative position.

We pass on now to the consideration of the special varieties of tumors. Inasmuch as this is a work for practical surgeons, I have not ventured to adopt Adami's classification, though I believe it to be the most scientific that has hitherto been devised. It involves, however, the use of a new terminology, and

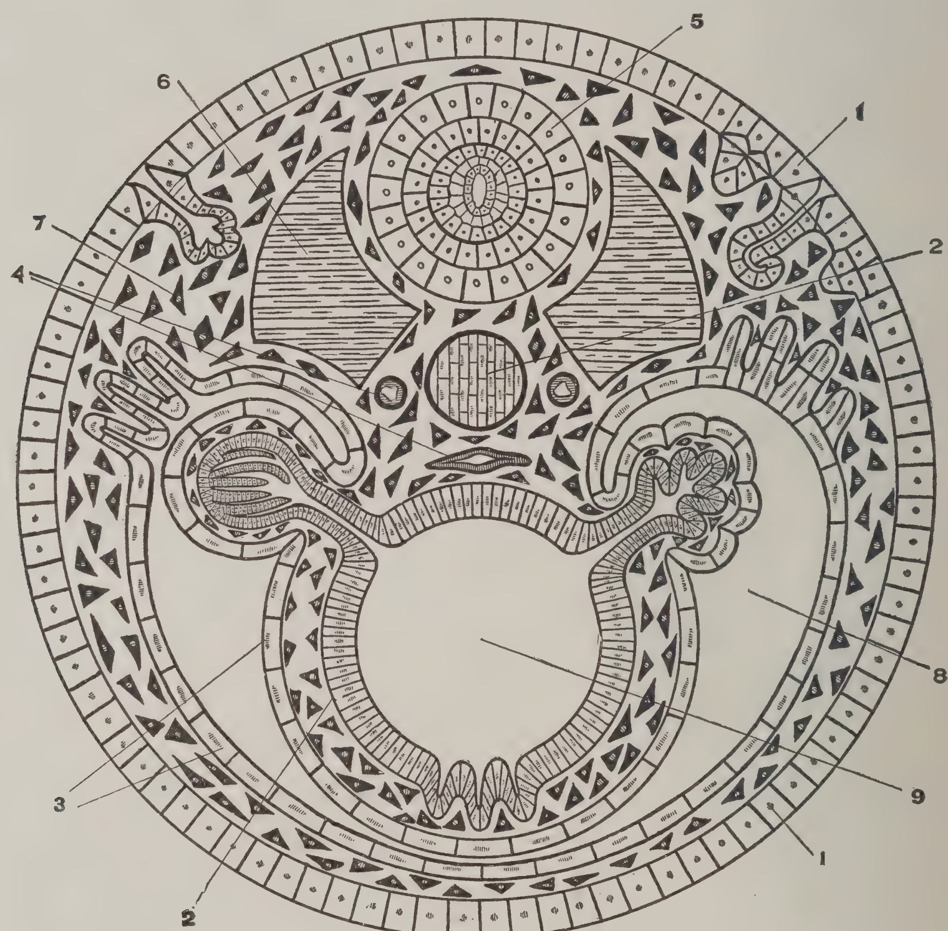

FIG. 84.—Scheme of Tissue Relationships. (Adami.)

Lepidic tissues: 1, Epiblast (ectoderm and glands); 2, hypoblast (entoderm and glands); 2′, notochord (hypoblast); 3, mesothelium (lining body-cavity), with derived glands; 4, endothelium (lining vessels).

Hylic tissues: 5, Epiblastic (forming nervous tissues); 6, mesothelial (forming striated muscles); 7, mesenchyme; 8, pleuro-peritoneal cavity; 9, lumen of alimentary canal.

until this becomes generally understood any other course would be liable to create confusion. Nevertheless, in view of the importance of the subject, I have introduced as alternative designations the terms employed in this latest attempt at the classification of new growths on embryological principles. By a reference to the schemata given above, the subject will be made sufficiently plain.

I. TUMORS OF NON-EPITHELIAL TYPE.

These may be benign or malignant. For the most part they are mesoblastic and mesenchymatous, though notable exceptions occur. Histologically, they manifest this important peculiarity, namely, that the component cells are embedded in an intercellular matrix, and vessels penetrate between many of the cells. In general it may be said that they consist of connective tissue or its homologues.

The following tumors come under this category:

Benign.	Fibroma.	Typical mesohylomata.
	Myoma.	
	Lipoma.	
	Chondroma.	
	Osteoma.	
	Leiomyoma.	
	Rhabdomyoma.	
	Angioma.	Partly hylic and partly lepidic.
	Glioma.	Typical epihylomata.
	Neuroma.	
	Papilloma.	Typical epilepidoma.
Malignant.	Sarcoma.	Atypical mesohyloma.
	Endothelioma (perithelioma).	
	(*a*) Of blood-vessels and lymphatics.	Atypical mesolepidoma.
	(*b*) Of serous membranes.	Endothelial lepidoma.

Fibromata.

A fibroma is a tumor composed in the main of fibrous connective tissue. It contains, however, more or less numerous blood-vessels and, under certain circumstances, nerves or other structures.

Fibromata may arise from any tissue or organ, provided that it contain connective tissue. We find them, therefore, in the skin, fascia, the sheaths of nerves, periosteum, tendons, the mamma, and uterus, less often in the ovary, bladder, and intestinal tract. They are among the commonest of tumors.

In general terms it may be said that they consist of nucleated fibrous cells, of adult or nearly adult type, held together by a fibrillar matrix. Several subvarieties can be recognized, according to certain minor differences in their structure.

Being benign they do not, of course, form metastases, but not infrequently enormous numbers of separate tumors may be found scattered over the body. This is particularly the case with fibromata of the skin and nerve sheaths.

To macroscopic appearance, fibromata may assume the form of warty, nodular, papillomatous, sessile, or diffuse growths. On section, the denser forms are hard, grating under the knife, white, and glistening, with a fibrillated structure

suggesting the appearance of watered ribbon. The softer varieties are more homogeneous, sometimes semitranslucent, and the fibrillated structure is not so evident. The larger ones are not invariably uniform in texture, but may contain harder or softer areas, patches of œdema or gelatinous transformation, or of calcareous infiltration (*fibroma petrificum*).

Microscopically, the harder forms (*fibroma durum*) consist of a dense feltwork of coarse interlacing fibrils, among which can be seen somewhat scanty, elongated, spindle-shaped nuclei. Should the tumor have been œdematous, the fibrils are more or less dissociated and the cells to some extent hydropic. The softer forms (*fibroma molle*) are much more cellular; the nuclei are more abundant, plumper, and the fibrils are more delicate and aggregated into smaller bundles (Fig. 85). The fibrils are never arranged in regular parallel rows or

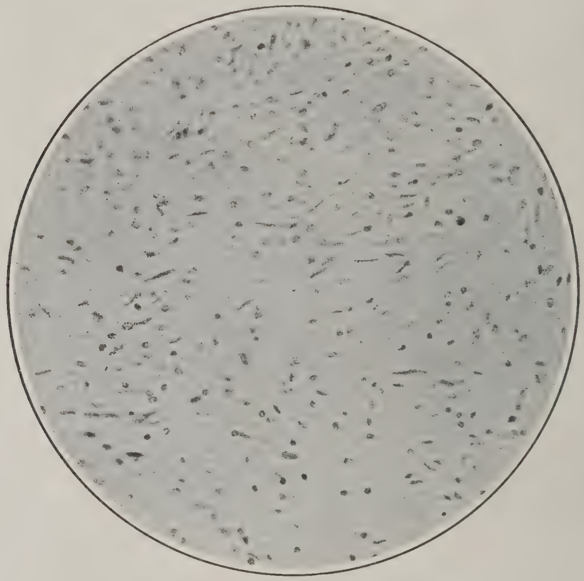

Fig. 85.—Soft Fibroma. Winckel No. 6, without ocular. (From the author's collection.)

layers, but interlace freely, and the various bundles may lie in planes that intersect one another at various angles. Not infrequently, too, the fibres are clustered in whorls, generally about some blood-vessel, duct, or gland tubule.

Inasmuch as fibromata are composed of proliferating fibrous tissue, we would naturally expect to find, and in fact this is not uncommonly the case, that in certain parts there are cells which are not quite so mature as those forming the great bulk of the tumor, round or stellate cells, and cells much shorter and plumper than the ordinary attenuated fibrous spindles. This peculiarity, when at all marked, often indicates a transition to a more cellular (sarcomatous) condition. The transformation of the young tumor cells into the adult type seems to take place after the same fashion as the normal proliferation of fibrous tissue. Fibromata are not always pure, but may be associated with the formation of adi-

pose tissue (*fibroma lipomatodes*), or may exhibit in parts a metaplasia into cartilage or bone (*fibroma ossificum*).

The vascularity of fibromata varies greatly. The blood-vessels may be scanty, large, and numerous (*fibroma teleangiectaticum*), or dilated into sinuses (*fibroma cavernosum*). Similarly, the lymph channels may be abundant (*fibroma lymphangiectaticum*).

Retrogressive changes are not common. The most frequent is infiltration with lime salts. Occasionally, we find fatty degeneration, liquefaction-necrosis, or ulceration.

There are certain forms of fibromata that deserve more than a passing mention.

Pedunculated fibromata are of somewhat frequent occurrence. They may be found in almost any part of the skin, but generally arise from the external (female) genitalia, the buttocks, thighs, or shoulders. When of any size, they are attached to the part by a narrow pedicle, and in time, owing to impaired nutrition, may undergo necrosis.

The so-called "fibroid" of the uterus is almost invariably a mixed tumor, consisting of a variable proportion of fibrous tissue and unstriped muscle (*myo-fibroma*). When approximating to the myomatous type, the tumor is soft, vascular, and presents a reddish, flesh-like appearance. When more fibrous, the growth is harder and much paler in color. Many cases can hardly, if at all, be distinguished by their gross appearance from simple fibromata.

Uterine fibroids are usually multiple and vary greatly in size. They may be all but invisible or attain the size of a man's head or an even larger object. The largest fibromata that we meet with are those of the uterus. The larger ones are hard, rounded, and nodular, and on section present the grayish-white, glistening, fibrillated appearance of the hard fibromata. Often nodules or whorls of fibres can be made out. A blood-vessel can sometimes be detected in the centre of the smaller nodules. Occasionally, the vessels are numerous, dilated, or sinus-like (*teleangiectatic* and *cavernous myo-fibromata*).

Microscopically, both muscular and fibrous elements can be detected in varying proportions (Fig. 93). The connective tissue tends to be aggregated about the blood-vessels. In many cases the tumor consists of little else but interlacing fibrils of fibrous tissue, forming nodules, strands, and whorls.

In order of frequency, myo-fibromata arise from the posterior wall of the corpus uteri; next, from the anterior wall; and lastly, from the fundus.

According to the site, it is usual to recognize four types: (1) the *intramural* or *interstitial*, (2) the *subserous*, (3) the *submucous*, and (4) the *intraligamentous*.

The subserous myo-fibromata, when pedunculated, may give rise to grave symptoms. Owing to interference with the circulation they may become inflamed, infarcted, necrotic, or gangrenous. By their size fibromata may lead to compression of the uterus and other important structures.

The etiology of uterine fibroids is still obscure. They do not occur before the age of puberty and are most common in elderly women. The condition is said to be more frequent in the black races.

Fibroma of the *breast* is perhaps the commonest form of new growth found in that organ. It is hardly ever met with in the male sex. It generally occurs during the period of active sexual life, but exceptionally may be found at puberty or after the menopause. Some cases arise without obvious cause, but many are traceable to some previous diseased condition of the mamma, such as mastitis or abscess. According to Thoma, the condition is hereditary in the female members of some families.

Fibroma of the mamma occurs under two main forms: as ill-defined strands of connective tissue intersecting the organ (*fibroma diffusum*), and as a rounded nodular mass (*fibroma tuberosum*). Nodular fibromata are usually multiple, and one or more of them may greatly exceed the others in size. It is important for the surgeon to bear in mind this tendency to multiplicity, as it has an important bearing on prognosis. The existence of other minute fibromata and the tendency to a diffuse fibromatosis of the supporting stroma of the breast explain the subsequent appearance of fibrous tumors after the operative removal of the more obtrusive growth (pseudo-recurrence).

As in the case of other tumors of this class, mammary fibromata are rarely pure. Proliferating glandular elements are almost invariably present to a greater or less extent. When this feature is marked the tumor can properly be termed an *adeno-fibroma*. Occasionally, these glandular structures are dilated into cysts—*cystadeno-fibroma*—into which fibrous processes covered with epithelium may project—*papilliferous cystadeno-fibroma*.

Microscopically, the nodular fibromata of the breast are composed of dense fibrous tissue in which are to be found more or less abundantly developed glandular ducts and acini. In one form the fibrous tissue is laid down in dense rings about the glandular structures (*pericanalicular fibroma*); in other cases growth seems to be so excessive that papillomatous outgrowths force their way into the gland spaces, which they dilate and eventually fill up. These processes are covered with glandular epithelium (*intracanalicular fibroma*).

Nodular fibromata are usually situated in the upper half of the breast near the periphery. They form firm, well-defined, rounded or oval masses, lying a short distance below the skin or in the depth of the breast. On palpation they are tense, so that it may be difficult to determine whether they are cystic or not without tapping, but may be definitely fluctuating in parts. They are often irregularly nodular. The overlying skin is not attached to the growth, nor are the regional lymph nodes enlarged. In the intracanalicular variety there may be a serous discharge from the nipple. A capsule is usually formed so that the growth is freely movable. The rate of growth is slow and the tumor rarely attains a large size. The cystic form, which is apt to be found in the older patients, grows

somewhat more rapidly than the other. Fibromata of the breast usually come into evidence for the first time at the puerperium or during menstruation, owing to the discomfort that they occasion at such times.

The peripheral nerves are not infrequently the site of multiple primary fibromata, which arise from the endoneurium. The tumors appear as spindle-shaped thickenings of the nerve bundles and may be so extensive that the nerve trunk undergoes a diffuse or irregular nodular thickening. Microscopically, these fibromata are of the soft type. In the case of the smaller spindles, the nerve fibres can easily be traced passing through the tumor, but in the larger ones they can be detected only at the points of entrance and exit of the nerves. As the physiological function is only slightly, if at all, impaired, it is probable that the axis cylinders are not destroyed. A new formation of nerve fibres, such as has been claimed by Klebs and others, cannot be said to be as yet substantiated, so that the name *neuro-fibroma*, sometimes applied to these tumors, is incorrect.

In one type of the affection the nerve trunks are the seat of diffuse fibrous thickening, so that large, subcutaneous tumors, composed of thick, wormlike cords, closely intertwined, are formed (*plexiform fibroma; Rankenneurom*).

The multiple soft fibromata found in the skin, constituting the affection known as *molluscum fibrosum*, have been shown by Von Recklinghausen to be fibromata formed on the peripheral portions of the cutaneous nerves.

It may, perhaps, be mentioned here that one variety of *elephantiasis* is due to a diffuse and extensive fibromatosis of the subcutaneous tissue similar in nature to the forms just described.

A very curious and somewhat rare form of fibroma is the so-called *keloid*. Some cases arise without obvious cause, "*idiopathic*" or *spontaneous* keloid; others are very definitely to be connected with some previous injury, *secondary*, *scar*, *cicatricial*, or *spurious* keloid. It is not impossible, however, that cases of supposed idiopathic keloid ought to be attributed to some previous injury which has left no noticeable trace, or has been so slight as to have been forgotten. Certain families and certain races, particularly the negro, are believed to be specially predisposed to this form of new growth.

Scar keloid often follows operations, burns, and slight cutaneous injuries. It has been known to occur after blistering, in the scars of leech bites, after piercing for earrings, after shaving, and in the scars left by various eruptive diseases.

Keloid may be met with in the form of oval nodules, elongated cylindrical growths, or as cordlike elevations, bands, ridges, or radiating processes. Keloid growths are usually single, but may be multiple. The tumor is firm, elastic, elevated above the general surface, and sharply defined. It is adherent to the skin, and the cutis covering it is thin, smooth, and of a whitish or pinkish color.

Histologically, keloid is a fibroma originating in the corium and, it is believed, from the fibrous adventitia of the vessels of the corium. The older portions of the growth are of the type of the *fibroma durum*, but in the younger parts the

tumor is more cellular. The various layers of the corium, papillæ and rete pegs, remain intact.

Keloid tends to progress for a number of years, when it may become stationary. It rarely involutes spontaneously.

Myxomata.

Myxomata are tumors of mucoid character. Structurally, they are composed of cells floating in a homogeneous, semifluid, mucinous matrix. The cells are mononuclear, bipolar or stellate, and provided with more or less elongated protoplasmic processes which interlace freely (Fig. 86). The intercellular substance varies in amount in different tumors and in different parts of the same tumor.

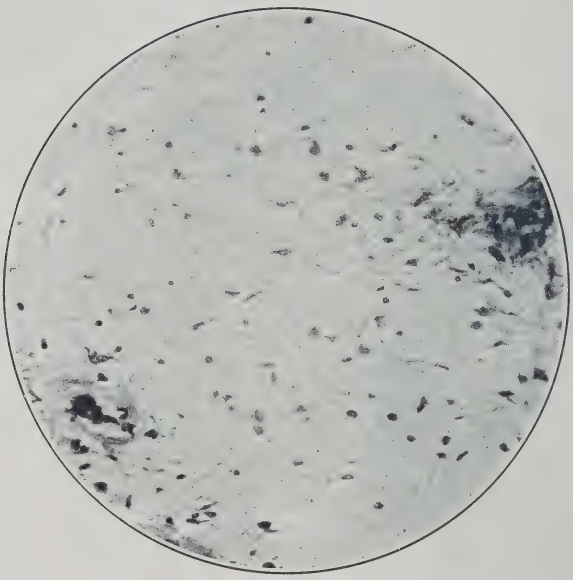

Fig. 86.—Myxoma. Winckel No. 6, without ocular. (From the author's collection.)

When abundant it gives a characteristic gelatinous, semifluid, somewhat translucent appearance to the growth. Myxomata are grayish or pinkish-gray in color, owing to the presence of blood-vessels that are more or less distinctly visible in the substance. On section, a jelly-like or ropy substance—mucin—exudes, which is not soluble in water and gives a whitish precipitate when treated with alcohol or dilute acetic acid.

Myxomata are rarely pure in type, but are usually combined with other tissues of a homologous nature, such as fibrous tissue (*fibro-myxoma*), fat (*myxoma lipomatodes; lipo-myxoma*), or cartilage (*chondro-myxoma*).

Mucoid tissue is closely related to fibrous tissue. The truth of this is evident when we remember that in the fœtus the fibrous and fatty tissues are first blocked out in mucoid material. Consequently, myxomata are commonly met with in the same regions from which fibromata and lipomata also spring. Further, mucoid tissue occurs in the adult body only in the vitreous humor of the eye, from which

structure myxomata never develop. Myxomata, therefore, always exhibit a certain deviation from the tissues in which they are found. In other words, they are heterologous within narrow limits. The embryonic character of the cells also explains the well-known instability of the tumor, which, though classed among the benign growths, has a tendency to take on malignant action, the transformation occurring in the direction of sarcoma (*myxo-sarcoma*). True myxomata have to be distinguished on the one hand from tumors, such as fibromata, sarcomata, and carcinomata, that have undergone secondary mucinous degeneration, and, on the other, from growths that, owing to vascular disturbances, have become œdematous. In both cases the resemblance may be striking.

Myxomata, fibro-myxomata, and lipo-myxomata may originate in the connective tissue of the skin, fascia, periosteum, mucous membranes, and muscle sheaths, in the subcutaneous and subserous fat, the bone marrow, and, occasionally, in the mamma, salivary glands, and testis.

Myxomata of the mucous membranes occur singly or as multiple primary tumors. The ordinary mucous polyp of the nose is a good example of these growths. Similar tumors are sometimes found in the larynx, intestinal tract, and uterus. They approximate in structure to pure myxomata and often contain more or less altered portions of mucous glands. Some of the cases can be traced with some probability to the irritation caused by a pre-existing catarrh of the mucous membrane affected, but this influence cannot always be effective.

The *hydatidiform mole* of the uterus is a myxomatous transformation of the chorionic villi (*myxoma chorii racemosum*).

Myxo-sarcomata, so called, are due either to a cellular transformation of a simple fibro-myxoma, or to the mucinous degeneration of a sarcoma. They behave as sarcomata and form metastases.

Lipomata.

Lipomata are tumors composed in the main of adipose tissue. They form soft or moderately firm growths, well defined, nodular, often lobulated (Fig. 87). They vary greatly in size. Those found in the internal viscera, such as the kidney, are often only discovered on microscopical examination, but those met with in the subcutaneous and retroperitoneal tissues may attain a notable size. The superficial ones, on palpation, are soft and yielding. On section, they are composed of masses of fat bounded and held together by fibrous septa.

Microscopically, the fat cells bear a general resemblance to those of the subcutaneous tissues, but are larger (Fig. 88). Not infrequently there is a considerable admixture of other elements, such as fibrous tissue (*fibro-lipoma*), mucoid tissue (*myxo-lipoma*). The blood-vessels vary greatly in numbers and size.

The lipoma is ordinarily a benign tumor. The sarcomatous transformation which is occasionally seen can only occur after the metaplasia of the cells of the tumor into fibrous or myxomatous tissue.

Lipomata may occur in the new-born, as, for example, the tumors that develop at or near the fissure in cases of spina bifida, but are much more common in later life They are found most frequently starting from the subcutaneous tissue of the back, buttock, thigh, abdominal wall, throat, and axilla, less often from the intermuscular connective tissue, the subserous fat of the peritoneal cavity, the kidneys, mamma, the hands and fingers, under the aponeurosis of the frontal region, and in the meninges. They originate usually from pre-existing adipose tissue, but can develop from fibrous tissue containing no fat, as, for instance, the submucosa of the intestines. Masses of newly formed fat occasionally form in the epicardium and around the kidneys, especially in alcoholics and those suffering from interstitial nephritis, and are sometimes called lipomata. Before,

Fig. 87.—Lipoma from the Subcutaneous Tissues of the Gluteal Region. (Pathological Museum of McGill University.)

however, we can be sure that such accumulations of fat are true tumors we must be certain that they possess independent powers of proliferation. The autonomous nature of true lipomata is proved by the fact that when, from any cause, the general fat of the body is disappearing, the tumor itself remains unaffected.

Lipomata grow slowly and produce no effects save those of pressure. They are sharply bounded, do not infiltrate, and never form metastases. Like the fibromata they are not infrequently multiple, and, curiously enough, in some instances are symmetrically distributed. When of large size they are liable to be the seat of degenerative processes, calcification, necrosis, gangrene, or ulceration. The ulcers produced are often very foul, owing to the presence of fatty acids. Unless of large size, lipomata cause little trouble, but occasionally surgical inter-

ference is called for to remove tumors that are situated in awkward places or are troublesome on account of their weight.

Perhaps the form of most interest and importance is the *retroperitoneal lipoma*, so called. This is a somewhat rare form of lipoma, but occurs sufficiently often to warrant a detailed description. It is met with usually in those about middle life or somewhat later, but exceptionally has been observed shortly after birth (Lauwers). The female sex is somewhat more often affected than the male. The growth begins in the radix mesenterii or perirenal fat, less often in the subserous fat lower down. The rate of growth is slow, two or three years usually elapsing before death takes place. Enlargement of the abdomen, often asymmetrical, gradually supervenes, but for a long time produces no unpleas-

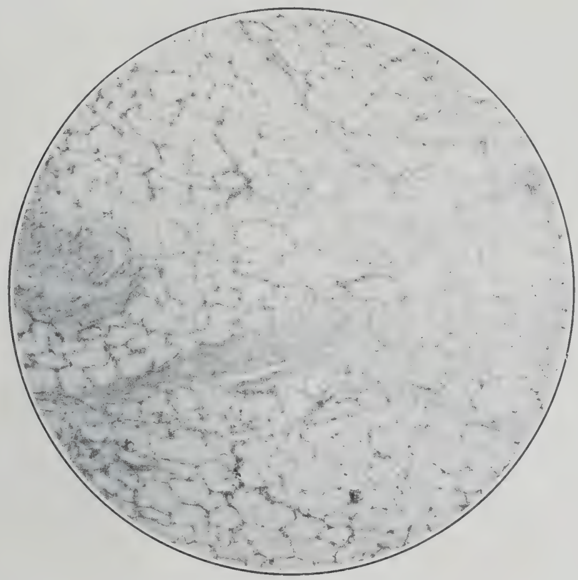

Fig. 88.—Lipoma. Winckel No. 3, without ocular. (From the author's collection.)

ant subjective symptoms. Some few subjects have suffered from vomiting, colic, flatulency, or other digestive disturbances. When, however, the growth becomes large it leads to pressure symptoms, intestinal obstruction, dyspnœa, and œdema of the lower extremities. In the later stages, weakness and emaciation are marked features. On examination, the abdomen is often enormously distended and gives the sensation of fluctuation. The percussion note is dull, but not infrequently a band of tympany can be traced across the front of the abdomen, due to the fact that the colon is pushed forward and lies over the tumor. Fluctuation is a characteristic feature and simulates so closely an accumulation of fluid that the erroneous diagnosis of ascites, ovarian cyst, and echinococcus cyst has been made. The insertion of a trocar readily demonstrates, however, the nature of the growth. A dry tap under these circumstances should always suggest the probability of a retroperitoneal lipoma being present.

The size which these tumors may attain is extraordinary. Adami (*Montreal Medical Journal*, January and February, 1897) has reported one approximating forty-five pounds in weight, and Waldeyer (*Virch. Arch.*, XXXII., 1865, p. 543) one of sixty-three pounds.

Histologically, they are rarely, if ever, pure lipomata. All sorts of combinations of fatty tissue with its homologues, fibrous tissue, mucoid tissue, cartilage, and bone, may occur. Perhaps the majority are of the nature of myxolipomata (*lipoma myxomatodes*). Deposits of calcareous salts may occur.

Such lipomata produce their effects by reason of their size and are not ordinarily malignant. In a case of Waldeyer's secondary growths developed in other parts. This occurrence is readily explained when we find that the larger lipomata of this type almost invariably present sarcomatous transformation in some part or other, a change which will usually be revealed on careful examnation.

The causes at work in the production of lipomata are somewhat obscure. Traumatism seems to be of some importance. Thus, tumors of this nature have been known to develop in an old scar. The rather frequent occurrence of lipomata about the shoulders has suggested that the pressure of clothing or suspenders may have something to do with it. The facts that lipomata are sometimes found in infants, especially those affected by spina bifida, and that adipose tissue is an important constituent of some teratomata, make it probable that a congenital vice of development is at work in some cases. This view is supported to some extent by the fact that certain lipomata of the kidney are traceable to aberrant suprarenal "rests." The symmetrical distribution of the growths in some instances has been explained on the basis of a neurotrophic disturbance.

In not a few cases, however, none of the influences mentioned can be traced.

Chondromata.

Tumors consisting in the main of cartilage are called *chondromata*. They vary considerably in size. The smaller ones are approximately spherical, while the larger are apt to be irregular, nodular, or lobulated. The major part of the growth is composed of hyaline, fibro-, or elastic cartilage, which is enclosed in a fibrous capsule carrying the nutrient vessels. In the larger cartilaginous tumors there are, in addition, numerous vascular fibrous trabeculæ dividing the cartilage into a number of islands.

Chondromata are of almost stony hardness, unless they are degenerated, and on section have a whitish, somewhat translucent appearance. The larger ones not infrequently show areas of softening or of calcification, and may contain cystic cavities filled with a gelatinous material.

Cartilaginous tumors commonly develop in situations where cartilage is normally present, but occasionally arise in structures that are devoid of cartilage, such as the parotid gland, the testis, mamma, submaxillary gland, and skin.

They originate in the proliferation of normal pre-existing cartilage cells (*ecchondromata*), or they may arise from other forms of connective tissue, especially fibrous tissue (*enchondromata*).

Histological examination reveals a certain amount of variation in structure. Not only may these differences exist between tumors, but between different parts of the same tumor. The cartilage is usually of the hyaline variety, less often elastic or fibrous. Even in hyaline chondromata there may be here and there areas of a more fibrous character, and at the periphery the cartilaginous structure merges gradually into the fibrous investing membrane. The cartilage cells proper vary considerably in numbers, size, shape, and arrangement. They may be large or small, rounded, spindle-shaped, or stellate. In some cases they are abundant, in other cases scanty. It is not uncommon to find evidences of retrogression such

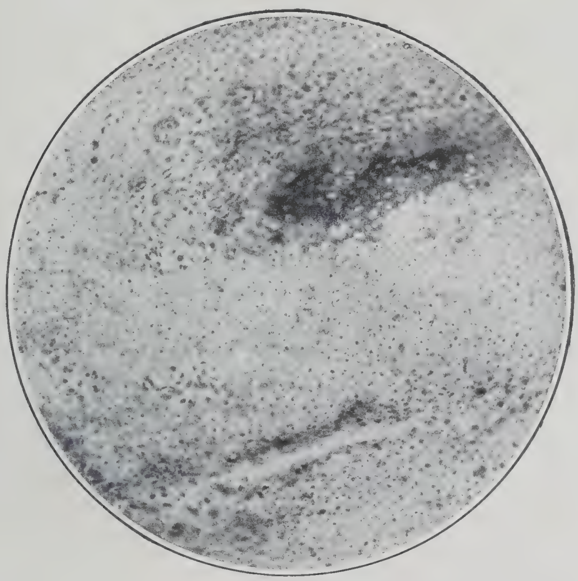

FIG. 89.—Chondroma from Mamma of Bitch. At one point the specimen shows a calcareous deposit. Winckel No. 3, without ocular. (From the author's collection.)

as fatty degeneration, mucinous degeneration, liquefaction, and calcification. In many instances we can recognize in parts myxomatous tissue (*myxo-chondroma*) or true bone (*osteo-chondroma*) (Fig. 89).

Ecchondromata take the form of rounded or polypoid outgrowths and are usually primarily multiple. They arise from the cartilage of the ribs, trachea, larynx, intervertebral discs, or elsewhere, and not infrequently present evidences of myxomatous change, petrifaction, or ossification. The *ecchondrosis physalifera* of Virchow, otherwise called *chordoma*, is a curious tumor about the size of a cherry, arising from the clivus Blumenbachii or the spheno-occipital synchondrosis. As it grows it pushes the dura before it, and, when the symphysis is ossified, appears as a rounded mass attached by a short pedicle to a small conical

elevation on the surface of the bone. It is firmly adherent to the dura. It used to be thought that this remarkable growth originated in the remains of the embryonic notochord, but this is now known to be incorrect.

Enchondromata usually occur in bones and originate in the periosteum or bone marrow, but never from the articular cartilages. In regard to situation they particularly affect, among the long bones, the phalanges of the hands and feet and the femur. The enchondromata of the phalanges are usually multiple primary growths, starting from different bones, and may appear during the early years of life. They may attain the size of the fist (Fig. 90). Among other parts of the skeleton the flat bones of the pelvis and shoulder girdle, and the maxillæ, are often

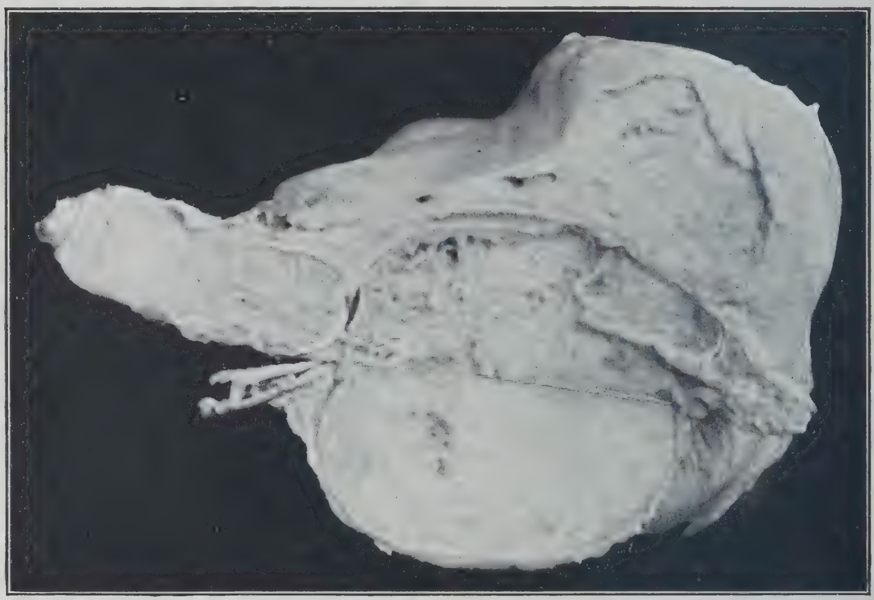

FIG. 90.—Chondroma of the Phalanges ; section made in an axial direction. (Pathological Museum of McGill University.)

involved. The enchondromata of the femur, pelvis, and shoulder girdle may grow to an enormous size.

Perhaps the most interesting of the enchondromata are those forms that arise in the parotid or testis, and in other structures that normally do not contain cartilage. As a rule they are of a mixed type. The mixed tumor of the parotid and submaxillary glands is composed of fibro-cartilage, containing more or less numerous irregular epithelial elements, remains of the original glandular acini which have become enclosed in the tumor. Connective tissue and myxomatous tissue are also commonly present, and there may be a cellular variation in the direction of sarcomatous or carcinomatous metamorphosis. The mixed tumors of the testis are similar except that they are as a rule composed of hyaline cartilage. These mixed growths are malignant and form metastases through the blood- or lymph-

channels, metastases which are sarcomatous or carcinomatous in character. It should not be forgotten, also, that apparently simple enchondromata may, on occasion, produce metastases. For this to occur it would seem that a preliminary mucinous degeneration of the tissue must take place. Multiple small, rounded, local, metastatic growths, varying in size from that of a bean to that of a walnut, are sometimes to be found among the muscles in the neighborhood of large enchondromata of the shoulder girdle, pelvis, and femur, which have become softened at the centre. Metastases are also occasionally found in the internal viscera, notably the lungs, and lymph nodes. To account for these, an ingrowth of the cartilage cells into the large veins has been observed (Virchow). This power of forming metastases is a somewhat variable one. Short of forming regular secondary nodules in distant parts, a somewhat rare event, we may have local metastasis, or simply a tendency on the part of the tumor to send in processes into the interstices of the tissues in the immediate neighborhood. At all events, the peculiarity is sufficiently well marked to stamp the enchondromata as relatively the most malignant of the organoid tumors.

With regard to the etiology of chondromata we are on somewhat firmer ground than we are in regard to some other tumors. Cartilaginous new formations may arise from pre-existing cartilage cells which have taken on excessive action, though we can no more explain the essential nature of this increased activity of growth than we can in the case of tumors generally. In the case of the enchondroma of bone, Virchow's explanation is commonly accepted, namely, that during the post-embryonic development of bone, islets of cartilage become displaced, owing probably to rachitic processes, and subsequently proliferate. The heterologous tumors of the parotid, salivary glands, testis, and skin are best to be explained as originating in misplaced embryonic "rests." Defective closure of the branchial clefts is an important factor in the case of the enchondromata of the salivary glands and the skin of the neck.

Apart from the rather rare event of the formation of metastases, chondromata produce their effects largely by pressure and by their size. Superficial chondromata may soften in the centre and discharge the liquefied material through the skin, so that a necrotic cavity is produced which subsequently suppurates. A chronic ulcer is thus formed which shows no tendency to heal and subjects the patient to the danger of general septic infection.

Osteomata.

Tumors composed of osseous tissue are called *osteomata*. Two main varieties are recognized, the *osteoma eburneum*, formed of compact bone, and the *osteoma spongiosum* or *medullary osteoma*, corresponding in structure to the spongy portion of normal bone. Besides these, there are certain new formations of bone, which are more or less doubtfully to be included in the category of true tumors, and to which special names are given. Circumscribed outgrowths on the external sur-

face of bones are termed *osteophytes*, or, if tumor-like, *exostoses* (cortical osteomata). Circumscribed outgrowths within the substance of bones are known as *enostoses* (central osteomata). *Hyperostosis* is a diffuse and generalized increase in the bulk of a bone.

Osteomata are single or multiple, and arise usually from bones and teeth, periosteum, and from the attachments of fasciæ, tendons, and ligaments (Fig. 91). Fascial, tendinous, and ligamentous osteomata may be firmly united to the bone (*continuous fascial, tendinous,* and *ligamentous osteomata*), or be separate from it and often movable (*discontinuous osteomata*). Exceptionally, osteomata originate from the soft tissues, such as the muscles, dura, choroid and sclerotic, penis.

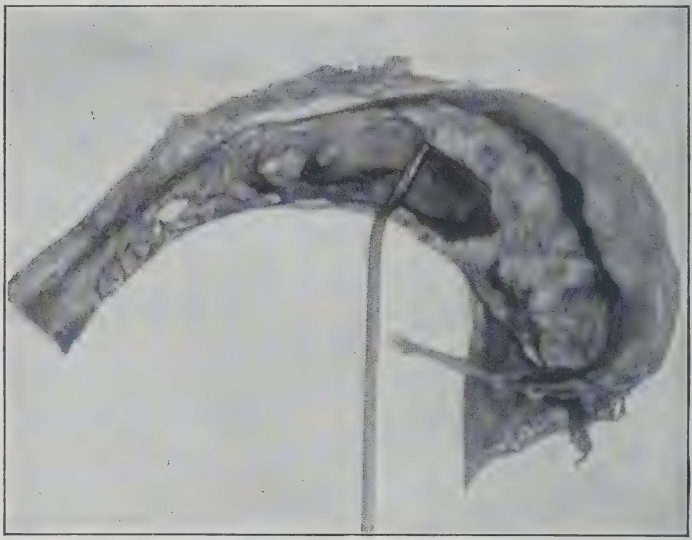

FIG. 91.—Osteoma in the Falx Cerebri. (Pathological Museum of McGill University.)

Heteroplastic osteomata are sometimes met with in the parotid, lungs, brain, diaphragm, skin, and tongue.

In appearance osteomata may be uniformly smooth, conical, rounded, or button-like, or, again, irregular, rough, and warty.

The hard osteomata, or osteomata eburnea, are of dense ivory-like consistence, and are composed of thick, compact bone, with relatively small nutritive canals, resembling the cortical substance of the long bones.

The spongy osteomata are similar to normal spongy bone, and are made up of delicate bony trabeculæ enclosing wide marrow spaces.

Dental osteomata originate in the cement of the tooth, which consists of true bony tissue. They start from the root of the tooth, where they form masses varying in size. The *odontoma* is composed of dentin and occurs not only on the root, but also on the neck and crown of the tooth. It is to be attributed to some disturbance of the pulp during the development of the tooth.

The osteoid tumors, growths closely allied to the osteomata, and, in fact, forming a subvariety of them, deserve mention. They arise from the periosteum, chiefly from that of the larger long bones, such as the humerus and femur. They are composed of osteoid tissue, which differs from bony tissue only in the fact that it is devoid of calcareous salts. These tumors have been confused with the cartilaginous growths, and have been termed osteoid chondromata, but are distinguished from them by the fact that they contain numerous Haversian canals carrying the nutrient vessels.

The histogenetic development of osteomata is interesting. In some cases the formation of the new bony tissue takes place after the fashion of normal bone, that is to say, through the agency of osteoblasts, or by a process of metaplasia. In this way bony tumors can arise either from the bone marrow or from the periosteum. The proliferating cells may give rise, in the first instance, to cartilage, which subsequently is converted into bone (*cartilaginous exostosis*), or the bone may arise directly from connective tissue (*connective-tissue exostosis*). The hard osteomata of the calvarium are probably to be referred to excessive local subperiosteal osteogenesis. The irregular, flattened plates and spicules of bone or osteoid substance which are found in the dura of the brain and cord have been explained on the basis of a reversion of the dura to its more primitive osteogenetic function.

The multiple osteomata bear certain close analogies to the multiple chondromata. Many of them occur during the active period of growth, and are connected with the articular surfaces of bones, suggesting some anomaly of development. A hereditary tendency has been traced in some cases (Heymann, Nasse); and rickets, as in the case of the chondromata, may play an important rôle. Bessel-Hagen and Nasse draw attention to the fact that persons the subjects of multiple osteomata often present other disturbances of development in the skeleton. The heteroplastic osteomata, such as those found in the parotid and tongue, are to be attributed to misplaced cells or embryonic "rests." Finally, osteomata may apparently originate in the proliferation of cellular elements of unknown character.

Besides the forms above mentioned, there are a number of others that can hardly be included among the tumors, inasmuch as they are not autonomous new formations. Such are many osteophytes, hyperostoses, and exostoses, certain of the discontinuous osteomata, the bony plates that sometimes form in the choroid and sclerotic, and the irregular masses of bone found in muscle. In many cases irritation of some kind, or inflammation, appears to be the predisposing cause, though it seems likely that such can only act in the presence of some inherited tendency to cell proliferation. Of this nature are the so-called "riders' bone" and "exercise bone" that sometimes develop in the adductors of the femora and in the deltoid, and, possibly, also the extensive and progressive ossification of the connective tissue of the muscle in the curious disease known as *myositis ossificans*.

Myomata.

Myomata are tumors consisting chiefly of muscle fibres. Two varieties can be recognized, those composed of striated muscle, *rhabdomyoma* (myoma striocellulare), and those formed of unstriped muscle, *leiomyoma* (myoma lævicellulare).

The rhabdomyoma (von Zenker) is a somewhat rare form, the peculiar feature of which is that it is composed of more or less embryonic or undifferentiated striated-muscle cells. The cells of more adult type occur as multinucleated ribbon-like masses of protoplasm of varying thickness, presenting well-marked transverse striation and sometimes also longitudinal fibrillation. The more immature cells are in various stages of differentiation. There are round cells, pos-

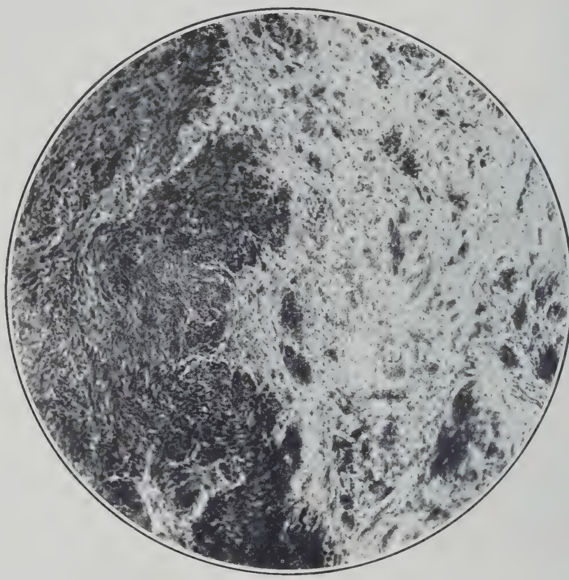

Fig. 92.—Fibro-myoma (" Fibroid ") of the Uterus. The darker tissue consists of unstriped muscle; the lighter, of fibrous tissue. Winckel No. 3, without ocular. (From the author's collection.)

sessing no special peculiarities, that are scarcely, if at all, to be distinguished from the earliest forms of connective-tissue cells; irregularly rounded or oval cells, presenting radial or concentric striations; spindle cells having long processes, with or without a faint striation; and small ribbon-like masses of protoplasm without striæ. The bands and spindles are aggregated into bundles and interlace more or less freely.

To gross appearance, there is nothing specially characteristic of the rhabdomyomata. They form nodular growths, and, if on a free surface, may have a papillomatous or polypoid arrangement.

It is important to recognize the fact that the rhabdomyoma is a tumor of embryonic type. Its cells always fail in attaining perfect maturity. Consequently, we ought to class these growths along with the myxomata and chondro-

mata as tumors of unstable character, tending to be malignant. The presence in rhabdomyomata of comparatively undifferentiated round and spindle cells has led some to fall into the error of regarding these as immature connective-tissue elements, and calling such growths *rhabdomyo-sarcomata.* It is more natural to suppose that the cells in question are simply striated-muscle cells in an embryonic condition, though it cannot be denied that a sarcomatous variation of the connective tissue of rhabdomyomata does occasionally take place.

Rhabdomyomata may arise in structures normally containing striated-muscle fibres, but are usually heterologous. In more than half the cases the growth originates in the kidney or kidney pelvis; less often it is in the testicle or uterus; rarely it has been found in the vagina, urinary bladder, the voluntary muscles, the wall of the heart, subcutaneous tissue, mediastinum, œsophagus, stomach, pa-

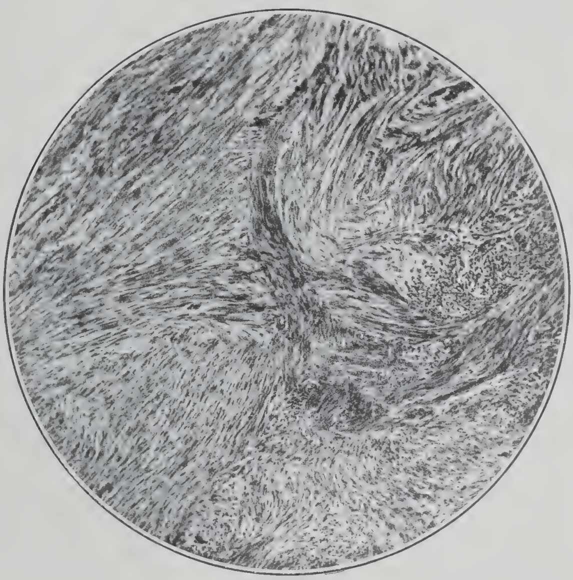

FIG. 93.—Myoma from the Arm. Winckel No. 3, without ocular. (From the author's collection.)

rotid, and orbit. Newly formed striated-muscle fibres may also be recognized in that peculiar congenital condition known as *macroglossia.*

The facts that the majority of these tumors are found in situations devoid normally of muscle, where the various infoldings of the different germ layers are very complicated, and that they occur frequently at birth or shortly after, suggest that they arise from misplaced muscle "rests" derived from the primitive myotomes. In certain teratoid tumors striated muscle is found combined with cartilage, bone, and epithelial elements.

Much more common are the tumors composed of smooth-muscle fibres. They are found most often in the uterus and prostate, occasionally in the alimentary tract and urinary passages, more rarely in the skin and subcutaneous tissues. Structurally, they are composed of smooth muscle fibres, arranged in bundles which intersect one another in different directions, embedded in more or less vas-

cular connective tissue. According to the predominance of one or other of these elements we may recognize a *myoma fibrosum* or *fibromyoma,* a *myoma teleangiectaticum* or *cavernosum,* or a pure myoma, *myoma molle.* Not infrequently the connective-tissue elements are so abundant as to warrant us in calling the tumor a *myo-fibroma.* The majority of the uterine myomata, so-called, are of this type. In fact, in America at least, they are more properly classed with the fibromata than with the myomata. (Fig. 92.)

Myomata and fibro-myomata form irregular or warty-looking tumors, which on section present a variable appearance according to their nature. The fibrous

Fig. 94.—Myomatous Enlargement of the Prostate, obstruction to the outflow of urine caused by the overgrowth of the so-called middle lobe; consecutive hypertrophy of the wall of the bladder. (Pathological Museum of McGill University.

portion is of fibrillated texture, firm, and of a shining white color; the muscular part is pinkish or bright grayish-red.

Histologically, the muscle fibres may be recognized by the fact that they are long spindles arranged somewhat regularly into bundles, and possess elongated rod-shaped nuclei (Fig. 93). The character of the cells can usually be ascertained on macerating some fresh material for from twenty to thirty minutes in from three

to four per cent. caustic-potash solution and then teasing it out with needles. Degenerative changes, fatty degeneration, softening, cyst formation, and calcification are not uncommon events in leiomyomata. Occasionally, the muscle fibres may atrophy, thus converting the tumor into a simple fibroma.

Leiomyomata are benign tumors and develop during adult or advanced life. Very exceptionally leiomyomata have been observed during the first few years of life.

With regard to their mode of origin, it may be said in general terms that they may arise wherever there is unstriped muscle. In the case of uterine myomata and certain myomata of the skin, the muscle bundles can often be made out to be arranged around the vessels, the ramifications of which they more or less follow, suggesting that the growth has originated from the muscular walls of these vessels. In other parts of the body, myomata are also sometimes found in the walls of the blood-vessels, particularly the veins. Some of the myomata of the skin have been shown to be derived from the arrectores pilorum. Now and then epithelial elements, in the form of cell masses or imperfect acini, have been found in uterine myomata. They have been variously explained as being remnants of the Muellerian or Gaertner's ducts, or of the Wolffian body. Possibly, they are only portions of the uterine mucosa which have been pinched off and have become embedded in the tumor.

The clinical results produced by myomata depend mainly upon their size and position. Myomata of the uterus lead to distortion and displacement of that organ, and to pressure or tension on the other pelvic viscera. Myomata of the prostate often start from the so-called middle lobe, and may, by encroaching upon the urinary passage at the neck of the bladder, result in obstruction to the outflow of urine with all that this condition implies. (Fig. 94.) Multiple myomata of the skin are occasionally very painful (*tubercula dolorosa*).

In rare instances, leiomyomata undergo sarcomatous transformation and give rise to metastases.

Angiomata.

Under this caption are included a number of tumors and tumor-like formations the chief peculiarity of which is that they are composed mainly of vascular channels, either blood or lymph vessels. These vessels may be newly formed, or may consist of the pre-existing vessels of the part more or less altered, either in the direction of enlarged calibre, or in that of increase in length or hypertrophy of their walls. Tumors consisting mainly of blood-vessels are termed *hæmangiomata*, or *angiomata*, this term being used in a restricted sense; those composed of lymph channels are known as *lymphangiomata*.

A hæmangioma consists of arteries, capillaries, and veins, which are supported and held together by connective tissue or by tissues homologous with it, such as adipose and mucoid tissue. We can, therefore, recognize mixed forms of vascular tumors, such as *teleangiectatic fibroma, lipoma, myxoma.* When we

have a cellular variation in the direction of malignancy, we may speak of *angiosarcoma.*

According to the character of the vessels that go to make up the tumor, it is customary to distinguish several subvarieties.

In *hæmangioma simplex* (hæmangioma teleangiectaticum; teleangiectasia) there is an excessive development of capillaries, with a relatively scanty formation of arteries and veins.

Hæmangioma arteriale (tumor vasculosus arterialis) is mainly composed of small arteries, with a relatively small proportion of capillaries and veins.

Hæmangioma cavernosum (cavernoma; tumor cavernosus) presents numerous large vascular spaces or sinuses, lined with endothelium, resembling the structure found normally in the corpora cavernosa of the penis.

The best known example of the simple hæmangioma is the *nævus vasculosus,* one of the forms of "birth-mark." This is found commonly in the skin and is present at birth, though it may attain its greatest development somewhat later. In these cases we cannot always speak of the condition as a true tumor, for the vascular area may be badly defined, without any elevation of the skin, but in other instances the area is well localized, penetrating into the subcutaneous tissues, associated with undoubted new formation of fibrous tissue, and covered with hypertrophic epithelium. In still other cases regular warts or flattened tubercles are formed.

The simple, smooth nævus appears in the skin as a bright red (*nævus flammeus*) or bluish-red patch (nævus vinosus: "port-wine stain"). At the periphery, many smaller vascular spots may often be seen. The red color is due to the presence of numerous wide and dilated vessels filled with blood, situated partly in the corium and partly in the subcutaneous fatty tissue. Occasionally, similar formations are met with in other structures, such as the mamma, liver, bones, brain, and spinal cord. The abnormality consists mainly in a circumscribed dilatation of pre-existing or newly formed capillaries. The dilatation may be spindle-shaped or cylindrical, or, again, saccular, or all three. The dilated vessels may be separated from one another by normally constituted capillaries or by capillaries only slightly dilated. The vascular walls are usually thin, or at any rate not specially thickened.

In the variety of simple hæmangioma known as the *angioma simplex hypertrophicum* the capillaries are exceedingly numerous and held together by relatively little connective tissue. The lumina of the vessels are only moderately dilated and the walls are thick and cellular, resembling arterial walls. In a few cases it happens that the endothelial cells proliferate and thereby encroach on the lumina. The tumor is divided into lobules by connective tissue in such a way that each lobule is made up of a highly convoluted tangle of capillaries with thickened walls. Here and there atrophied remains of sweat glands may be sometimes detected.

The *hæmangioma venosum* is composed chiefly of veins, the capillaries being only slightly if at all enlarged. The dilatation of the veins is cylindrical, ampulliform, or saccular. The vascular walls are sometimes thickened.

The *hæmangioma arteriale* consists of numerous small arteries, with a comparatively small development of veins and capillaries. A curious variety of this is the *angioma arteriale racemosum* (cirsoid aneurysm; angioma arteriale plexiforme; Rankenangiom). Here the arteries of a particular district, such as the forehead or scalp, are dilated, thickened, and highly convoluted. The tumor feels on palpation somewhat like a bag of worms. The blood can usually be squeezed out of the vessels, but they quickly fill up again so soon as the pressure is removed. A bruit can usually be heard over the affected area.

The *cavernoma* (tumor cavernosus) consists of large, irregularly shaped sinuses, lined with endothelium, and separated one from the other by a more or less cellular connective tissue. The various blood spaces may here and there be seen to communicate with one another.

Cavernomata are found usually in the skin and subcutaneous tissues, but occasionally in the viscera, especially the liver (Fig. 95), more rarely in the kidney, spleen, uterus, intestine, bladder, muscles, and bones. In the skin they form bluish-red, somewhat elevated or warty elevations (*nævus prominens*), or may lead to a uniform and extensive enlargement of the part, constituting one form of *elephantiasis*.

The histogenesis of hæmangiomata is extremely interesting. Many of the cases are present at birth or appear during the earlier years of life, and are met with in situations corresponding to the embryonic lines of fusion, such as the facial and branchial clefts. The angiomata found at the orifices of the body, in the face, neck, and upper part of the breast, are of the nature of *fissural angiomata*. Certain of the nævi vasculosi, or "mother's marks," though not all, come under the category of fissural angiomata. Many of the angiomata belonging to this class are at first little more than teleangiectases, but, after a more or less prolonged period of latency, they may increase in size, and in time be transformed into large projecting masses, resembling a cock's comb.

Other hæmangiomata appear to have some connection with the nervous mechanism (*neuropathic angiomata*). Such are the teleangiectases which begin as small red spots on the skin and then gradually spread over the surface in an area corresponding to the peripheral distribution of some cutaneous nerve.

Certain multiple, nodular hæmangiomata are occasionally met with in old people, and are often termed *senile angiomata*. Thoma would place the cavernoma of the liver, which also occurs in advanced life, in this group.

Traumatism is an important factor in some cases. Of this nature are some cicatricial tumors or keloids which appear after injury and are particularly rich in vessels.

It must be admitted that the above classification of the angiomata is by no means complete, for many forms, and not the least important, such as certain vascular tumors of the skin, muscles, glands, and intestines, cannot be explained on the grounds mentioned. Possibly here congenital anomalies of development, though this is a simple conjecture, may have to be taken into account.

Thoma has drawn attention to the important part that physical and mechanical principles play in determining the origin and development of angiomata. This investigator has shown that the new formation of capillaries is related to the blood-pressure within the capillaries of a part and to the condition of the surrounding tissues. The normal intravascular pressure is dependent on the strength of the cardiac impulse and the resistance of the extravascular tissues. An increase of pressure so that it exceeds the normal will result in the production

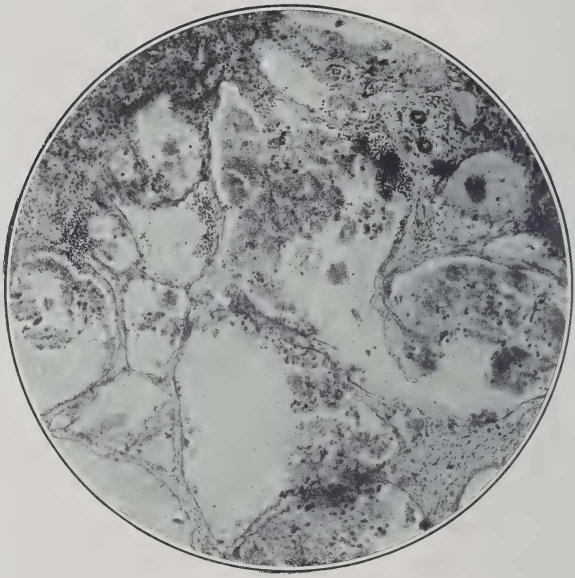

Fig. 95.—Cavernous Angioma of the Liver. Winckel No. 3, without ocular. Normal liver tissue is shown above and to the left. The fibrous trabeculæ of the blood tumor are well seen. (From the author's collection.)

of new capillaries. This may be explained as an attempt on the part of the tissues to establish an equilibrium. Disturbances of the blood pressure, as can readily be understood, might easily be brought about by errors of development and derangements of the vasomotor mechanism. Further changes are dependent on the rate of the blood flow. If the rate be under the normal, capillaries with narrow lumina are formed ; if it be greater, the capillaries will become more or less dilated. The rise in blood pressure, which must occur at some time in the process, will lead, in accordance with well-known pathological principles, to increase in the thickness of the capillary and lesser arterial walls.

The *lymphangioma,* or *angioma lymphaticum,* is in most respects analogous to the hæmangioma, save that the vascular spaces contained therein are lymph

channels instead of blood-vessels. The supporting stroma in which the vessels are embedded may be fibrous, fatty, or mucinous.

Three anatomical forms are recognized: the *lymphangioma simplex* or *telean-giectasia lymphatica*, the *lymphangioma cavernosum*, and the *lymphangioma cys-toides*.

In simple lymphangioma the lymphatic vessels in a more or less circum-scribed area are dilated and their walls thickened. In the cavernous variety the lymph channels are very numerous and much dilated, so that the structure, on section, has a somewhat spongy texture. The supporting stroma is scanty, thin, delicate, and transparent. In the last-mentioned form cysts varying in size from that of a pea to that of a walnut or larger may be produced.

As in the case of the hæmangiomata, developmental anomalies are of great etiological moment. Many of the lymphangiomata are found in connection with the sutures and fissures of the body. Of this nature are the lymphangiectasias met with in the tongue (*macroglossia*), gums, lips (*macrocheilia*), neck (*hygroma colli congenitum*), skin (*nævus lymphaticus*), subcutaneous tissues, and vulva. A diffuse cavernous dilatation of the lymphatic channels of the skin or subcuta-neous tissues, as, for instance, in the thigh and scrotum, gives rise to one form of elephantiasis. Some of the lymphangiectases of the skin, subcutaneous tissues, peritoneum, and mesentery, appear late in life and are acquired rather than con-genital. Lymphangiomata of the mesentery contain chyle and, hence, are some-times called *chylangiomata*. Cystic lymphangiomata are sometimes met with in the peritoneum, but are rare. Some authorities would class certain of the pig-mented nævi, pigment patches, freckles, and fleshy warts with the lymphangi-omata.

Lymphangiomata in the course of their growth may extend widely and dis-locate or enclose portions of the neighboring tissues. They may reach the surface of the body and there discharge, forming lymph fistulæ and causing lymphor-rhœa.

Both the hæmangiomata and the lymphangiomata are to be classed with the benign growths. As, however, they contain newly-formed endothelial elements and connective tissue, we occasionally find that they take on malignant action, becoming extremely cellular (*perithelial* or *endothelial angiosarcoma*).

Lymphangiomata may attain a considerable size, and the cavernous and cystic forms are often multiple. On section, these tumors exude lymph, which may be clear, or cloudy from admixture with lymph corpuscles or blood. The contents of the cysts may be fluid, or, again, partially or completely coagulated.

Histologically, the lymph spaces are lined with endothelium and held to-gether by a rather cellular fibrous tissue. Almost any tissue may, however, at times, enter into the composition of the stroma. Here and there in the support-ing substance collections of lymphoid cells may be found.

Gliomata.

Gliomata are tumors derived from the neuroglia or supporting stroma of the central nervous system. They are limited, therefore, to the brain and cord, and to those parts of the peripheral nervous system which represent prolongations of the primitive cerebral vesicles, namely, the retina, optic nerve, and olfactory bulbs.

Gliomata may be single or multiple, and vary considerably in size, though they never become very large. As a rule they resemble more or less closely the nervous tissue in which they are found, and indeed cases are not infrequently met with where we are only able to infer the presence of a tumor from the fact that there is a local swelling of the brain substance and that the normal distinctions between the various parts of the organ are obliterated.

On section through a glioma we find that it is usually badly defined, infiltrating the surrounding tissues; it is sometimes grayish in color, moderately firm and somewhat translucent, resembling the normal gray matter of the central nervous system; it may be grayish-white, rather dense and hard; or, again, it may be grayish-red or dark red, owing to the presence of numerous vessels. Hemorrhage into the substance of the tumor, fatty degeneration, softening, and necrosis are not infrequent accompaniments.

Histologically, a typical glioma is composed of a meshwork of delicate refractile fibrils, among which can be seen embedded more or less numerous rounded or oval nuclei. These nuclei, on closer inspection, are found to be surrounded by a small quantity of cell protoplasm (Fig. 96). On macerating the tissue and teasing it out with needles the cells referred to can be shown to be bipolar and stellate in shape and to possess shorter or longer, sometimes branching, processes. The blood-vessels are often abundant and may be dilated (*glioma teleangiectaticum*). The relative proportions of cells and fibrils vary in different cases. Some gliomata are cellular (*glioma molle*), others are more fibrous (*glioma durum*).

We can better understand the histogenesis of the gliomata if we remember the way in which the neuroglia normally develops. The glia, like the specific nerve elements, is of ectodermic origin, being derived from the undifferentiated epiblastic cells heaped up about the primitive dorsal groove. These cells eventually are separated from the superficial ectoderm and become aggregated about a central space, the neural canal, which is lined with cells that permanently retain their epithelial type. While certain cells undergo marked differentiation, and eventually are converted into the highly complicated nerve structures, others remain more primitive and assume many of the characteristics of connective tissue. The latter, the glia cells, originate, both in the highest and lowest vertebrates, in the ependyma cells, which are now known to belong to the supporting structures. In certain of the more primitive animals, such as the amphioxus, the supporting stroma is composed entirely of ependymal cells, but higher in the scale we find that the principal part is taken by stellate cells (astrocytes). In

the case of mammals it is believed by some that the astrocytes are not derived directly from the ependymal cells, but from intermediate forms, which may be termed astroblasts. All glia cells, whether provided with long or short processes, brush cells or stellate cells. are therefore, ultimately derived from the same precursors.

It has usually been taught that the various processes are closely related to the cell bodies, being, in fact, protoplasmic prolongations of the latter, an opinion based upon studies conducted with the Golgi method of staining More recent methods, notably those of Weigert, Mallory, and Beneke, have proved, however, that this is not altogether correct. According to Weigert, the cells of human

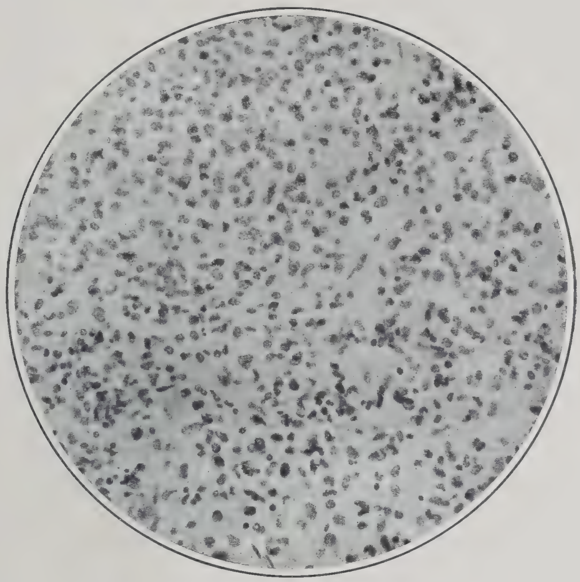

Fig. 96.—Glioma, from Cerebral Cortex. Winckel No. 6, without ocular. (From the author's collection.)

neuroglia possess protoplasmic processes only during embryonic life. Adult neuroglia is made up of cells and fibrils, the latter greatly predominating.

Bearing these facts in mind we are able to get a more adequate conception of the various forms of gliomata that we actually meet with. Thus, we have: (1) The glioma durum, or fibrillary form, corresponding to mature glial tissue; (2) the astrocytic glioma, composed of Deiters' spider and brush cells; (3) a highly cellular form, resembling a small round-celled sarcoma, possibly derived from a still more undifferentiated type of cell, namely, the astroblast; (4) gliomata composed of cells of ependymal type. It must be admitted, however, that while the more recent methods of investigation have proved of great value in differentiating the various forms of glioma one from the other, in another direction they have perhaps proved more confusing than helpful, for the relationship of such conditions as sclerosis of the central nervous system, nodular gliosis, and the central gliosis of syringomyelia remains still quite obscure.

A word or two with regard to the gliomata of the retina may not be out of place. These tumors are met with only in childhood, usually in the earlier years. One or both eyes may be attacked, and more than one member of a family may be affected with the disease. The tumor may originate in any of the layers of the retina, but usually in the deeper parts. It grows more or less rapidly, extends forward into the vitreous humor and invades the uveal region, or it may perforate the sclerotic posteriorly and attack the orbit and brain. Eventually, the whole eye is destroyed and we have a large vascular, fungating mass which projects externally and leads to destruction of the neighboring soft tissues and bone. Secondary growths are formed in the regional lymph nodes and in distant organs. Histologically, we may recognize several varieties, which are strictly in accord with the classification of the other gliomata indicated above. The most common form is a highly cellular growth, composed of undifferentiated cells, resembling closely the small round-celled sarcoma. Less often the growth is composed of closely aggregated cells with processes, or astrocytes. In other cases we find curious rosette-like formations, so that the tumor resembles in some degree the cells of the layers of rods and cones. The layer of rods and cones corresponds histogenetically with the epithelial cells lining the central neural canal, and, therefore, is to be regarded as of ependymal nature. Flexner, therefore, would term such tumors *ependymal gliomata.* In consideration of their histological appearance and the physiological function of the cells from which they are derived, they are often called *neuro-epitheliomata.*

The relationship of glioma to sarcoma is at present a somewhat debatable question. The fact that gliomata are embryologically of ectodermic origin, while sarcomata are mesodermic, would of itself suffice to indicate that there are fundamental differences between these two forms of new growth. It is a fact, however, that the vessels in gliomata are provided with sheaths of mesoblastic fibrous tissue, and it is, therefore, theoretically possible that a cellular variation of this mesoblastic structure might on occasion give rise to a true sarcomatous neoplasia. Such tumors would, therefore, be mixed in character, consisting both of newly formed glial and sarcomatous elements. As we have seen, however, certain gliomata are derived from relatively undifferentiated glial cells, and their resemblance to round-celled sarcomata is so close that we are often at a loss to make the differential diagnosis. This being the case, we are hardly justified, in my opinion, in speaking of new growths of this histological type as "glio-sarcomata," even in view of the fact that they are often malignant. When we consider that tumors of this type are composed of relatively undifferentiated cells, cells which according to well-known pathological principles must be endowed with great proliferative capacity, it is not surprising that they at times take on excessive and aberrant action. This view is supported by what we have already learned in connection with other tumors composed of relatively immature cells, such as the soft fibroma, myxoma, and chondroma, which, as we have seen, may

occasionally produce both local and distant metastases. We shall, I think, be more logical if, for the time being at least, we speak of the forms in question as "malignant gliomata," until microscopical investigation shall have proved beyond cavil that the round cells present therein are derived from the mesoblastic vessel sheaths.

Some interesting points come up, too, in regard to the ependymal gliomata. It cannot now be denied that we occasionally meet with tumors composed of cells resembling somewhat closely cells of ependymal type. Flexner, for example, records a brain tumor composed of cells resembling for the most part the ependymal cells found in the embryonic human cord. These cells were arranged in a radial fashion around the blood-vessels, toward which their processes were directed. The processes came together at a point somewhat short of the vessel wall, so that a small space existed between them and the wall. It is conceivable that more fully developed or adult ependymal cells might be competent to give rise to tumors. One point to which attention should be directed is that the cells of the ependymal gliomata may assume more or less perfectly an epithelioid type. In cases where the cells are of this character, or are spindle-shaped, especially if they be grouped about the vessels, the resemblance to the endotheliomata and peritheliomata is striking. With regard, therefore, to tumors originating in portions of the brain containing ependymal elements, as for instance the pituitary, which resemble endothelial and perithelial formations, it would be well always to consider the possibility of their being glial and ependymal in nature.

It is quite possible that the gliosis occurring in the condition known as syringomyelia, which has proved such a puzzle to investigators, may be explained on the lines indicated above. Flexner (*Journal of Nervous and Mental Disease*, May, 1898) mentions having seen a case of syringomyelia in which the tumor mass was composed largely, if not entirely, of cells of an early ependymal type.

In view of the fact that our ideas in regard to gliosis and gliomatosis are in a transition stage, and that our ignorance on many important points is not slight, it is not surprising that but little is to be said on the subject of the remote etiology of these growths. Certain of the ependymal gliomata, and the gliomata met with in childhood, are probably to be referred to some developmental anomaly or aberration. Trauma has been held by some to play a part. Possibly, also, toxic and infectious agents will be found to be of some etiological importance.

Gliomata produce their effects in accordance with their size and position. Those in the central portion of the brain give rise to no clinical symptoms save those of pressure. Others, when situated in areas functionally important, both by pressure and destructive infiltration damage the neurons and interfere proportionately with the origination and conduction of impulses. Thus we may get muscular paralysis, disturbances of sensation, interference with muscular tone, pain, and paræsthesiæ.

Neuromata.

The term *neuroma* is used somewhat loosely by surgeons to designate almost any tumor arising in connection with nerves. In this category would come the so-called "amputation neuroma," multiple cutaneous neuromata, and the plexiform neuroma. To which may possibly be added the neuroma or neuro-glioma ganglionare. All these forms have this in common, that they consist of nerve cells or fibres held together by a fibrous or neuroglial matrix. It should be remarked, however, that in the strict sense of the term the word "neuroma" should be applied only to growths consisting wholly or in part of newly formed nerve elements. Of course it is difficult in many cases to decide, when nerve cells or fibres are discovered in a tumor, whether these are newly formed or not, and this is the whole point at issue between the pathologists. A large number of growths occupy debatable ground, but more careful study, connected with modern technique, has served greatly to circumscribe the class of nerve tumors, though it has undoubtedly proved that neuromata, in the true sense of the word, do exist.

The so-called *amputation neuroma* belongs to what has been called the *traumatic neuromata*. It is perhaps the most common form of the false neuromata. As its name implies, the condition is found in connection with amputation wounds. In some cases of this kind the ends of the nerves within the stump are found to be swollen like clubs and firmly adherent to the cicatrix. Microscopic examination shows that these nodes are composed of medullated and non-medullated nerve fibres, irregularly interlacing, embedded in a dense scar tissue. Properly, such growths should not be classed with the neuromata, for they represent simply the ordinary process of regeneration modified by an unusual physical condition. The dense scar prevents the nerve fibres from growing straight forward in the axis of the nerve trunk, so that they become diverted and bend and interlace in a confused manner. The new formation of fibres is, therefore, not autonomous. Traumatic neuromata are occasionally met with in connection with injuries other than amputation. Division or compression of a nerve sometimes results in the formation of nodules composed of newly formed nerve fibres and connective tissue at the seat of injury.

An interesting class of cases is that which includes the multiple nodes sometimes found upon the peripheral nerves. These tumors are found not only on the trunk of the nerves, but also on their peripheral terminations and may affect a large part of the body, or, again, may be confined to a particular nerve district. Not uncommonly the tumors are situated in the skin where they form numerous, smaller or larger, usually soft nodules. They are often painful, owing to pressure upon the sensory fibres (*tubercula dolorosa*). Further investigation has shown that some of these cutaneous nodules are leiomyomata containing nerve fibrils, but the majority of them are to be regarded as fibromata (*q. v.*). The smallest nodules are only of microscopic size, but the larger ones may attain the size of a pea, a marble, or even a man's fist.

The so-called *plexiform neuroma* (Rankenneurom) has already been dealt with (see p. 305). Suffice it to say here that most authorities hold it to be simply a peculiar form of fibroma of the nerve sheaths, though a few good observers still maintain that the nerve fibres to be seen in this growth are newly formed, and that, therefore, the tumor is a true neuroma. Plexiform neuromata are found upon the head, trunk, and extremities, and lead to a condition resembling elephantiasis.

The multiple cutaneous neuromata found by Knauss in young children seem to be true neuromata. Here we find branching ganglion cells together with numerous medullated and non-medullated fibres, having no anatomical continuity with the nerves of the part.

"Neuromata" of the central nervous system have been described. In some of these, tumor-like masses of nervous substance have been found. Probably some of them are simply a misplacement or abnormal arrangement of the normal layers of the central nervous tissues, due to an anomaly of development. Or they may be merely artefacts, as Lubarsch has suggested. Inasmuch as certain of the forms just mentioned contain ganglion cells, they have been included with the neuro-glioma or neuroma ganglionare.

Ganglionic neuromata have been met with in the thoracic, lumbar, hypogastric, solar, and adrenal plexuses of the sympathetic system. Microscopically, they consist of a more or less dense glia-supporting stroma, in which can be seen irregularly distributed ganglia, and nerve fibres. They are probably true neuromata.

Papillomata.

Strictly speaking, the term "papilloma," as applied to tumors, refers to their external appearance rather than to peculiarities of histogenetic structure. Any tumor, in fact, which projects above the general surface of the tissue in which it is found, and has a convoluted, villus-like appearance, may properly be termed a papilloma. Such tumors illustrate particularly well the "organoid" character supposed to appertain to the benign growths we have been describing.

Papillomata consist of a central core of vascular, connective, or mucoid tissue, covered with one or more layers of epithelium. They are found springing from the skin and mucous surfaces and occasionally from the interior of cysts and the ducts of glands. They are rounded, cylindrical, lobulated, or cauliflower-like in appearance, or highly convoluted and villus-like. They may be sessile and attached by a broad base, or, again, may have a relatively narrow pedicle. The nature of the epithelium with which they are covered varies somewhat, but conforms more or less closely to that of the part from which they arise (Fig. 97).

Many cutaneous warts belong to the class of papillomata, as do certain congenital excrescences on the surface of the body, papillary nævi.

Papillomata of the mucous surfaces are found especially in connection with

the larynx and trachea, the stomach and intestines, the urinary bladder, and the genitalia, as, for example, the penis, vulva, vagina, uterus, and Fallopian tubes.

One of the most important types is the papilloma of the bladder, which takes the form of a cauliflower-like growth, composed of an aggregation of numerous delicate branching papillæ. These papillæ are composed of a small amount of vascular connective tissue covered with cylindrical epithelium. Papillomata of the bladder are usually situated at the fundus of the organ and are often mul-

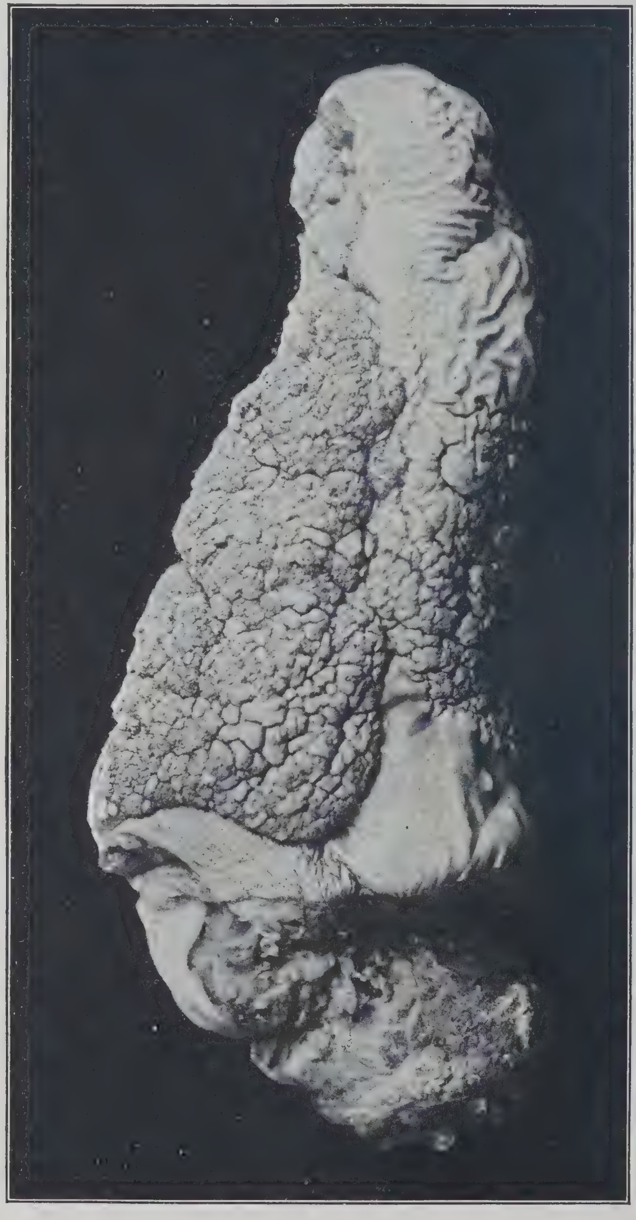

Fig. 97.—Extensive Papilloma of the Foot. (Pathological Museum of McGill University.)

tiple. They are of importance to the surgeon in that they frequently cause obstruction of the urine, with all that implies, give rise to hæmaturia, and may, occasionally, assume malignant action.

The most potent single factor in the causation of papillomata is irritation in its widest sense. Papillomata of the larynx are found in singers, public speakers, and others who strain the voice. Chronic congestion may be of importance here. Chronic catarrh accounts for many papillomata of the mucous surfaces, as, for example, "venereal warts" (condylomata acuminata). Many of the warts of the skin are properly to be referred to the effects of irritation. It is perhaps questionable, where chronic inflammation is the chief etiological factor, whether the new formation of tissue thereupon resulting should properly be classed with the autonomous new formations. It has probably more affinities with the simple, irritative hyperplasias. The papillary excrescences found in connection with many cystic adenomata may with much more reason be classed with true tumors.

Sarcomata (Atypical Meso-hylomata).

We have up to this point been considering a series of neoplasms, benign in character and of more or less perfect organoid type, which have this in common, that they reproduce the features of normal adult connective tissue, that is to say, fibrous tissue and its homologues. They are of mesoblastic origin, with the exception of the neuromata and gliomata, which are epiblastic, and the papillomata, which are partly mesoblastic and partly epiblastic.

Corresponding with most of these, and forming a cellular variation of them, we have another set of tumors, commonly known as *sarcomata.*

Sarcomata may be defined as malignant tumors of mesoblastic origin and connective-tissue type, having for the most part this peculiarity, that they are composed of cells that more or less completely fail to attain the morphological perfection of adult cells. These cells are immature or comparatively undifferentiated and are consequently endowed with great proliferative capacity. We find, therefore, as we might expect, that the sarcomata are the most malignant of tumors, that is to say, they grow rapidly, tend to recur locally after removal, form early and extensive metastases, and, finally, are apt to break down and ulcerate. The occurrence of local metastases accounts for the lobulated structure that so many sarcomata present.

Sarcomata arise from all forms of connective tissue and in any part where such structures are found. They develop, therefore, from fibrous tissue, fatty tissue, mucoid tissue, cartilage, and bone (Fig. 98). There are certain parts, however, where they are more common than elsewhere. They are met with oftener, for example, in the skin, fascia, intermuscular connective tissue, periosteum, bone, brain, and ovaries than in the lungs, liver, intestines, and uterus.

The gross anatomical appearance of sarcomata varies considerably according to circumstances. No one description applies to all. Many sarcomata, especially

those connected with bone and periosteum, attain a large size, others are almost microscopic. In color, they may be whitish, pinkish, or grayish-white, glistening and semitranslucent, at other times brownish, black, bluish, green, or slaty, from the deposit of pigment. In regard to consistence, some are soft, juicy, and brain-

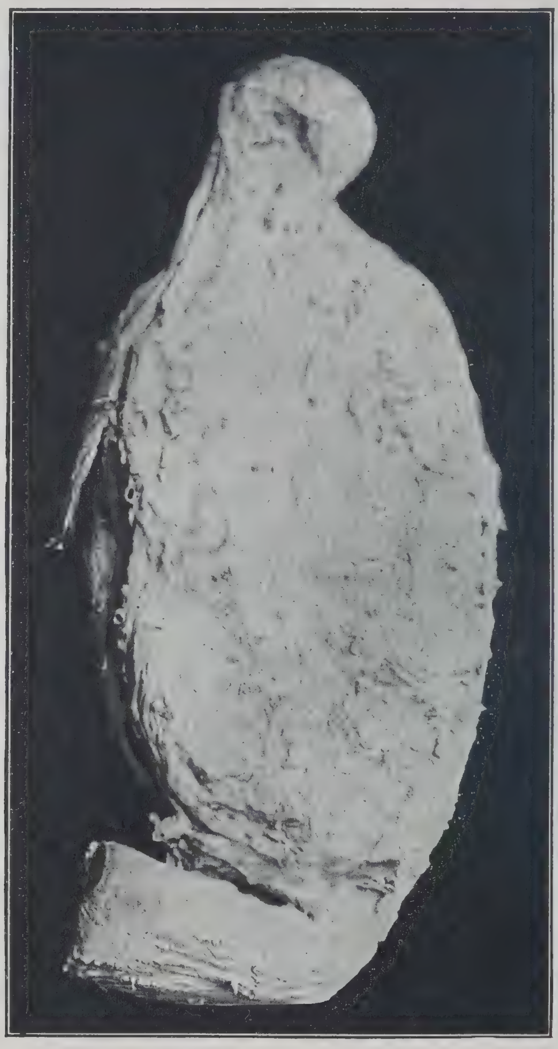

Fig. 98.—Sarcoma of the Shaft of the Humerus. (Pathological Museum of McGill University.)

like (*medullary sarcomata*); others, firmer, denser, and more fibrous; still others are of almost stony hardness.

Blood-vessels are more or less numerous, and may be dilated (*teleangiectatic sarcomata*). The vessels usually possess a regular wall, well defined from the tumor substance, but in some instances it is composed of the proper cells of the new growth.

Retrogressive changes, fatty and mucoid degeneration, hemorrhage, colliquative necrosis, caseation, gangrene, and ulceration are not uncommon.

As we have seen in the preceding pages, not a few of the benign, so-called organoid tumors may on occasion undergo at some point or other malignant, that is to say, sarcomatous, transformation. We may thus recognize, on the basis of etiology, *fibro-sarcomata, lipo-, myxo-, chondro-, osteo-, osteoid,* and *angio-sarcomata.* The sole exceptions are the neuromata and gliomata. The neuromata are excessively rare and not well understood, but so far as we know have no malignant cellular derivative. A malignant form of glioma is known, the so-called "glio-sarcoma," but we have elsewhere adduced reasons for thinking that the majority of these are not true sarcomata, unless we are prepared to use this term in the widest sense. Sarcomata may, however, arise directly from connective tissues without passing through the intermediate stage of benign neoplasia.

For descriptive purposes it is usual to classify the sarcomata according to the character and arrangement of the cells composing them. Perhaps the arrangement adopted by Ziegler is as convenient as any. He recognizes: (1) *Simple sarcomata,* tumors composed of a uniform aggregation of cells of connective-tissue type, but immature; (2) tumors which, owing to the peculiar arrangement and grouping of their component parts, more closely approximate the *organoid type,* in some cases resembling tumors of definitely epithelial type; and (3) tumors presenting *secondary changes* in their specific cells, stroma, or blood-vessels, changes that give them a peculiar and characteristic appearance.

Simple Sarcomata.—The simple sarcomata may be divided according to the shape of their cells into small and large round-celled sarcomata, small and large spindle-celled growths, and mixed forms. All gradations exist between the soft, highly cellular, and malignant medullary tumor and the more slowly growing, firm, fibrous sarcoma. At one end of the scale we have the small round-celled sarcoma, at the other the fibro-sarcoma and recurrent fibroma.

Small round-celled sarcomata are found more especially arising from the connective tissue of the locomotor apparatus and from connective-tissue stroma. They are met with also in the skin, lymph nodes, testis, and ovaries. They are soft and rapidly growing. On section, they are whitish or grayish-white in color, brainlike, and a milky juice can be scraped from the surface. Not infrequently they present necrotic, caseated, or softened areas.

Histologically, they consist almost entirely of round cells and blood-vessels. The round cells are small and delicate, with relatively little cytoplasm, and contain round or ovate, somewhat vesicular, nuclei. (Fig. 99.) Between the cells is a variable quantity of delicate granular and fibrillar stroma. It is usually quite scanty and may, indeed, be difficult to demonstrate. The vessels may be recognized as thin-walled channels coursing between the specific cells of the tumor. Here and there lymphoid cells can be made out whose nuclei stain more intensely than those of the tumor proper.

One particular form of small round-celled sarcoma deserves special remark. This is the so-called *lymphosarcoma.* It is very difficult to place this tumor, for pathologists are by no means agreed as to its nature. The enlarged lymph nodes in Hodgkin's disease are by some regarded as the result of a true autonomous neoplasia, thereupon termed lympho-sarcoma; others think that the condition is a simple inflammatory tissue hyperplasia. There is, undoubtedly, a new growth of the lymph nodes, which leads to local infiltration and the formation of distant metastases. It has the microscopical appearance of a small round-celled sarcoma (Fig. 100). Such a growth might arise from the connective tissue of the nodes (sarcoma of the lymph nodes) or by proliferation of the lymphoid elements (true lympho-sarcoma).

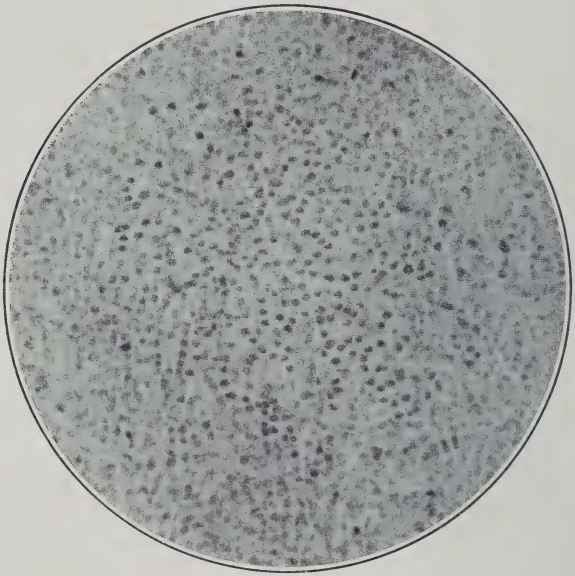

Fig. 99.—Small Round-Celled Sarcoma of the Cervix Uteri. Winckel No. 6, without ocular. (From the author's collection.)

Large round-celled sarcomata resemble closely the small-celled type and develop in the same situations. They are somewhat firmer and less malignant than the latter. The cells are larger, richer in cytoplasm, and possess one, two, or more large vesicular nuclei. Between the specific cells, and dividing them more or less definitely into groups or alveoli (*alveolar sarcoma*), there is a delicate fibrillated stroma containing here and there spindle and branching cells. The vessels are generally thin-walled.

Spindle-celled sarcomata are among the commonest forms of sarcomata. As a rule they are firmer than the round-celled form, and may appear on section even somewhat fibrous. Still, medullary forms occur. They are grayish or yellowish white in color, somewhat translucent, or, if vascular, may have a pinkish tinge. The cells lie for the most part side by side with their long axes pointing in the same

general direction. They may compose a large area of the tumor after this fashion, but are perhaps more commonly aggregated into bundles, which run in different directions, and, indeed, may to some extent interlace. Not infrequently there is a definite relationship to the vessels, the bundles being grouped about them after the manner of a sheath. On teasing out a sarcoma of this kind, the spindle cells composing it are found to assume various types according to the tumor. Some are oval or oat-shaped, others are short spindles, while still others are provided with long processes, so that they approximate closely to the type of the normal fibrous-tissue cell.

The supporting stroma is often scanty or may be scarcely, if it all, recognizable. In other cases it is more abundant and presents a fibrillar character. Those

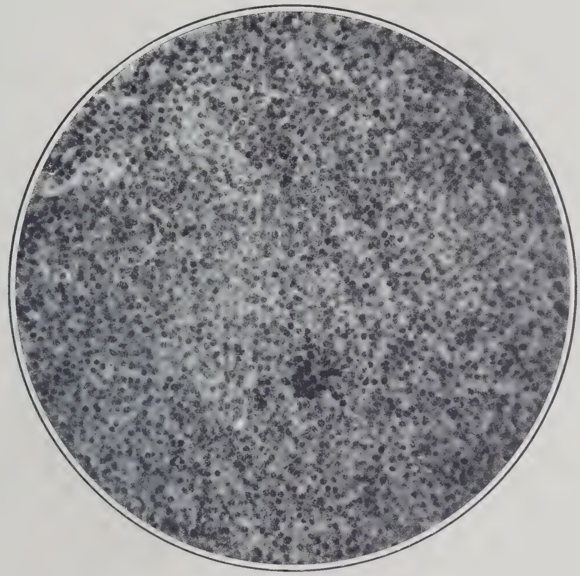

FIG. 100.—Lympho-sarcoma. Winckel No. 6, without ocular. (From the author's collection.)

spindle-celled growths which contain a relative abundance of stroma are usually termed *fibro-sarcomata* (Fig. 101).

The *mixed-celled sarcomata* are composed, as the name implies, of cells of several different types. It is not uncommon to find sarcomata, both of the round-celled and spindle-celled type, which on closer inspection are found to contain in addition oval, pyramidal, prismatic, stellate, or irregularly shaped cells. These cells may possess one or more nuclei. The most important variety is the *giant-celled sarcoma.*

This form is one of the most interesting to the surgeon, inasmuch as it develops in connection with the bones, occasionally in the breast, and may reach a great size. The shafts of the long bones and the alveolar process are the parts ordinarily attacked. The growth usually starts from the bone marrow, whence the term sometimes applied to it, *myeloid sarcoma* (Fig. 102), and in the course of its growth

leads to great rarefaction and destruction of the bone. The denser outer shell is thinned out and can be found over the surface of the tumor in the form of thin plates, that on palpation give a curious sensation like the crumpling of an egg shell (egg-shell crackle). In the alveolar process the giant-celled sarcoma forms one variety of the tumor known to surgeons as *epulis.* It is in this situation a dense, firm, sessile or nodular growth, tending to envelop the bone. It sometimes also originates in the antrum of Highmore. On section, giant-celled sarcomata are firm, somewhat fibrous, and frequently present a brick-red color from parenchymatous hemorrhage. The growth is one of the least malignant forms of the sarcomata.

Microscopically, there is usually a good deal of fibrous tissue here and there, so

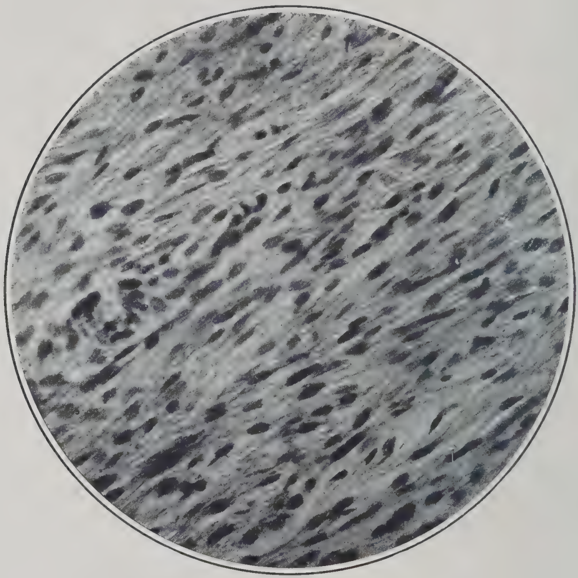

FIG. 101.—Spindle-celled Fibro-sarcoma. Winckel No. 6, without ocular. (From the author's collection.)

that the growth might be regarded as fibro-sarcoma. The specific cells are of mixed variety, round, oval, spindle, or irregular, but the characteristic feature is the presence of relatively enormous multinucleated cells. Small patches of hemorrhage can usually be made out in various parts (Fig. 103).

Sarcomata of Definitely Organoid Type.—In this group we place all those sarcomata that, from the peculiar arrangement of their cells, remind us somewhat of on organ. Thus, the cells may be aggregated into definite clusters or nests, surrounded by connective tissue (*alveolar sarcoma*); others have a tubular appearance not unlike that of a gland (*tubular sarcomata*); still others have a stratified appearance recalling the skin or a lining membrane. The type is not necessarily maintained throughout, it should be remarked. Thus a certain tumor may at one point present an organoid structure, while in other parts the appearance is rather

that of a simple, diffuse, round-, spindle-, or mixed-celled sarcoma. Again, some of the new growths coming under this category, consisting of large spindle, round, or cylindrical cells of epithelioid appearance, closely resemble the carcinomata, for which many of them have been mistaken, particularly if they have an alveolar ar-

FIG. 102.—Sarcoma of the Lower End of the Shaft of the Femur. The soft parts have been removed by maceration to show the rarefaction and expansion of the diaphysis. (Pathological Museum of McGill University.)

rangement. In not a few cases careful examination will show a gradual transition from a carcinomatoid to a definitely sarcomatous appearance in the same tumor. It is not surprising, therefore, that this class of tumors has led to much confusion of ideas and many erroneous deductions. Standing, as regards their histological appearance, on the border line between the carcinomata and the sarcomata, they have

been classed by different investigators in accordance with their individual bias as carcinomata or as sarcomata, while others have boldly met the difficulty by ignoring it and calling them sarco-carcinomata. I would like to emphasize here what I have said before, that it is far more scientific to go right to the root of the matter and classify these according to their origin and mode of development rather than on the basis of mere superficial resemblance. With careful study and the use of serial sections, the nature of these puzzling growths can usually be made out, though it may be freely admitted that the difficulties cannot always be cleared up.

The chief forms which we have to consider in this connection are the *angiosarcomata* and the *endotheliomata.*

Under the term angio-sarcoma we may include any highly vascular sarcoma.

Two main types may be recognized, though mixed forms and modifications occur, namely, the *angiomatous sarcoma* and the *perithelial sarcoma.*

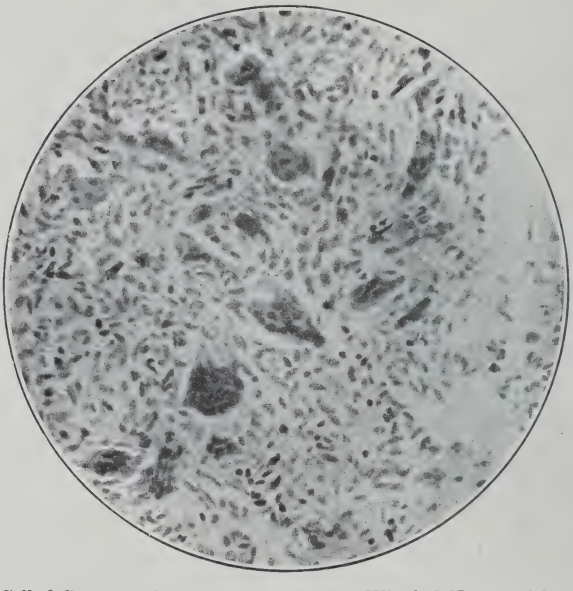

Fig. 103.—Giant-Celled Sarcoma, from the Periosteum. Winckel No. 6, without ocular. (From the author's collection.)

The first-mentioned may perhaps be regarded, at least in many cases, as a cellular variation of the angioma. It consists of numerous blood capillaries, between which are dense aggregations of sarcoma cells. The latter arise as a sarcomatous metamorphosis of the connective tissue forming the supporting stroma found in all angiomata. Owing to the abundance of the blood capillaries, such tumors often present a more or less distinctly alveolar arrangement, which may cause them to be mistaken for carcinomata.

In the perithelial sarcoma the structure is still more alveolar in appearance. The tumor is extremely vascular and the cells composing it are large, round, spindle-like, or cylindrical in shape; they are derived from the proliferation of the connective tissue or perithelium forming the adventitia of the vessels.

Thus, larger or smaller cell clusters are found grouped around the vessels, the intervening spaces being filled with connective or mucoid tissue, ordinary sarcoma cells, or a finely granular débris (Fig. 104).

The perithelial sarcomata are found in the kidneys, suprarenals, prostate, thyroid, parotid, and elsewhere. They are extremely malignant and tend to invade the veins of the neighboring parts. Numerous metastases may be formed. When the growths are superficial their extreme vascularity is manifested by rhythmical pulsation corresponding to the systole of the heart and by a blowing murmur on auscultation. The blood can often be squeezed out of the growth, only to return when the pressure is removed. Care should be taken not to confuse such

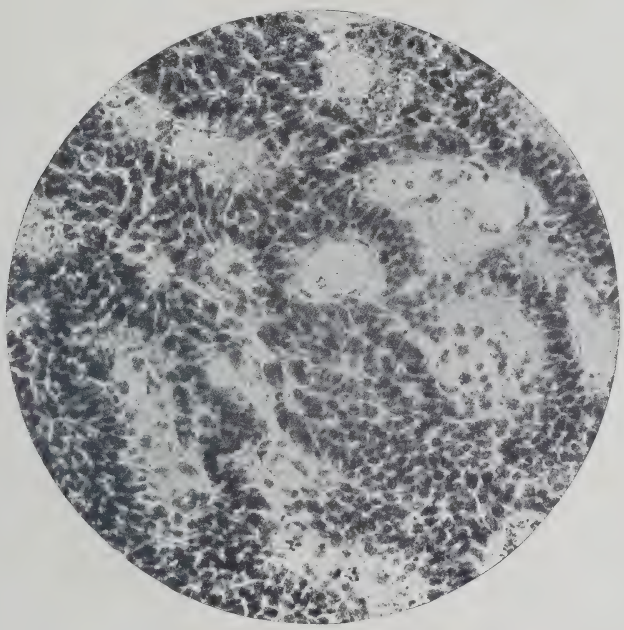

Fig. 104.—Perithelial Angio-sarcoma of the Pituitary Body. Leitz objective No. 7. (From the author's collection.)

angio-sarcomatous metastases with cirsoid aneurisms, arterio-venous aneurisms, or phlebectasiæ. While on the subject of angio-sarcomata I would point out that, as Flexner's case above referred to (p. 327) proves, there is a striking resemblance between the perithelial angio-sarcomata and certain tumors of the central nervous system, regarded as being probably ependymal gliomata, so that further study of brain tumors of this general type may result in considerable modification of our present ideas. Some new-growths, therefore, at present classed with the peritheliomata, may eventually turn out to be gliomatous in origin.

Under the term *endothelioma* we include all tumors derived from endothelial cells, whether of blood-vessels, lymphatics, perivascular lymph spaces, or of the larger serous cavities. The specific cells of such tumors are round, flattened, or cuboidal, and bear a strong general resemblance to epithelial cells. When we

remember this and also take into account the fact that the cells are not infrequently arranged in alveoli, bands, nests, and tubules, it is not surprising that certain of the endotheliomata should have been mistaken for carcinomata. The error is more likely to occur if only an isolated portion of the growth be examined; careful search, however, will often show at some point or other the direct continuity of the cells of the tumor with those of some lining membrane, and reveal the true nature of the growth.

The finer histological details of endotheliomata vary considerably according to the character of the structure from which they take their rise. In those originating in the endothelium lining blood-vessels, the specific tumor cells often show a definite relationship to the vessels, which may be so large, numerous, and tortuous as to give the tumor a highly complicated and peculiar structure (*angio-sarcoma plexiforme*). In parts it may be possible to detect the remains of the vessel walls which have been destroyed by the proliferating endothelium. Within the spaces formed by the more or less imperfectly formed tubules can sometimes be seen blood corpuscles, suggesting the origin of the growth from blood-vessels. Endotheliomata derived from the lymphatic channels present a very similar appearance. Those originating from the lining membranes of serous cavities and tissue spaces are apt to have a more alveolar character, clumps, nests, and anastomosing bands of epithelioid cells being embedded in a more or less abundant stroma of connective tissue. There may be but little ground-substance of a finely fibrillar character, it may form a well-defined stroma or, again, may be so dense and abundant that it gives a distinct scirrhous character to the growth.

The stroma in some cases exhibits peculiar secondary transformations. It may present mucinous degeneration, forming one variety of myxo-sarcoma; or there may be a hyaline change in the vessel walls and portions of the stroma, producing the curious growth known as the *sarcomatous cylindroma.*

Certain endotheliomata of the dura are intensely fibrous and contain laminated calcareous concretions, similar to those found normally in the pineal gland and meninges (brain-sand). They have, therefore, been termed *psammomata* (Fig. 105).

As will be gathered from the above remarks there is a striking similarity between certain of the more vascular endotheliomata and forms which I have already described under the group of *angio-sarcomata.* This has led to some confusion in the terminology. It would be well if, with Waldeyer, we should restrict the term *angio-sarcoma* to tumors originating from the adventitia of blood-vessels, and should include under *endothelioma* only such tumors as originate from endothelial lining membranes, whether vascular or not. It would be still more precise if we were to call tumors derived from the adventitia *perithelial angio-sarcomata* or *malignant peritheliomata.* The chief difficulty with this is that histologically the endotheliomata originating in the perivascular lymph spaces are almost identical with the perithelial angio-sarcomata. In the case of the latter growths it

ought to be possible to demonstrate the direct continuity of the cells nearest the blood-vessels with the adventitia, but this is often a matter of great difficulty.

Endotheliomata are found usually in connection with the serous sacs, the meninges of the brain and cord, and connective-tissue spaces, occasionally in the peribronchial connective tissue, the parotid, skin, and pituitary body. They form nodular or flattened sessile growths, tending to extend superficially. The denser, more fibrous forms, especially those of the meninges, are not particularly malignant, inasmuch as they infiltrate comparatively slowly and do not form distant metastases. The softer cellular forms are, however, often highly malignant.

The etiology is practically unknown. Irritation appears to play a part in some cases. I have met with two instances in which endotheliomata of the dura

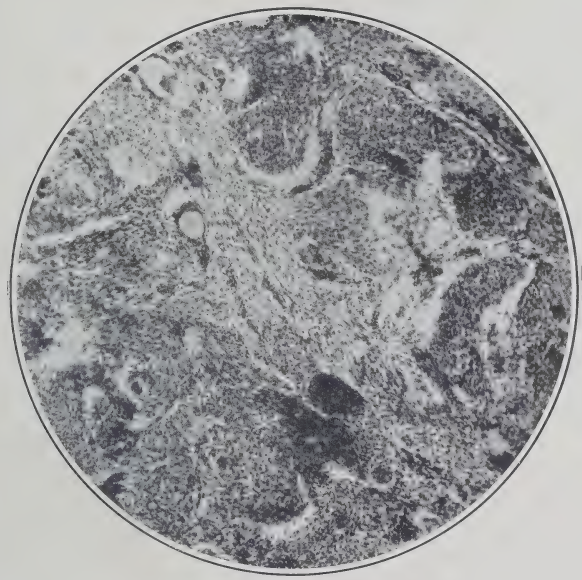

FIG. 105.—Endothelioma (Psammoma) of the Brain. Winckel No. 3, without ocular. (From the author's collection.)

mater appeared to be due to the influence of a sharp spur of bone projecting from the inner surface of the calvarium.

Sarcomata Presenting Peculiar Secondary Characteristics.—Under this heading we will discuss the pigmented sarcomata and certain forms presenting mucoid, hyaline, and calcareous transformation.

The *melanotic sarcoma* (melanosarcoma; melanoma; chromatophoroma) is a pigmented sarcoma, found usually in the uveal tract of the eye or in the skin. The growth varies in size and shape and is usually soft and friable. Its most striking feature is its color, which may range from yellow, brown, or gray to the most intense black. The coloration is not always uniform, and some forms may even be speckled. The vascularity may be great, and in that case it is not unusual to find areas of hemorrhage in the substance of the growth.

Melanotic sarcomata generally originate in structures that are normally pigmented, though there are occasional exceptions to this rule. Those found in the skin can usually be traced to pigmented nævi which have taken on aberrant growth. These growths are highly malignant and quickly produce metastases in various parts of the body, as the liver, lungs, intestines, muscles, bones, and skin. In some cases the secondary growths are exceedingly numerous, and often vary greatly in size, some being almost microscopical, others as large as a cherry or walnut. The primary tumor may be quite small and unobtrusive. Probably some of the cases reported as primary in the viscera are really secondary, the original growth having been overlooked.

Histologically, there are two types of melanotic sarcomata, the *spindle-celled* and the *alveolar*. The pigmented sarcomata that arise in the choroid of the eye are usually of the former variety, those occurring in the skin and in nævi are more apt to be alveolar.

The pigment is called melanin. Its nature is not thoroughly understood. The old idea was that it was derived from the coloring matter of the blood, but this is unlikely, as it contains no iron. It is known, moreover, to contain a considerable proportion of sulphur. Probably the composition of the pigment differs in different cases, and it is altogether likely that it is autochthonous in nature and produced by the metabolism of the chromatophores. The pigment takes the form of fine dust-like particles, granules, or lumps, both within the cells and in the interstices of the tissue (Fig. 106). It is highly refractile in appearance and of a yellowish, brownish, or black color. In the alveolar growths the pigment tends to accumulate in the cells at the periphery of the clusters and in the neighborhood of the blood-vessels. In the choroidal tumors the coloring matter is more uniformly distributed. The amount may be so great as to mask the true nature of the specific tumor cells. In fact it may so interfere with nutrition that liquefaction and necrosis result. It is a curious fact that the metastases often present a different degree of pigmentation from that presented by the original growth, and sometimes, indeed, they are quite colorless.

The exact status of these tumors is still not quite settled. Unna, Gilchrist, and others hold to the epiblastic nature of the forms arising in connection with pigmented nævi, while Ribbert contends that they arise from pigmented mesoblastic cells (chromatophores). On this point depends the question whether we are to class the melanomata with the carcinomata or with the sarcomata. In the case of the eye, the chromatophores, as Ribbert points out, are undoubtedly of mesoblastic origin.

Another pigmented growth of a somewhat remarkable nature is the *chloroma.* This is a rare tumor which develops in connection with periosteum, more especially that of the skull, vertebræ, and humerus. Its peculiar feature is its color, a green or greenish-yellow. This is most intense when the tumor is freshly cut, and fades somewhat on exposure to the air.

Histologically, the chloroma is composed of round cells, resembling large and small lymphocytes, held together by a delicate fibrous reticulum. The pigment occurs in the form of small, highly refractile granules within the cells, and is best made out in frozen sections or in teased-out material.

There is still much doubt in regard to the true nature of these tumors. Many hold them to be a form of lymph-adenoma and, therefore, related to leukæmia and Hodgkin's disease. The coloring matter gives some of the micro-chemical reactions of fat and is, probably, to be classed as a lipochrome.

Of the other secondary manifestations which sarcomata may undergo we may simply mention hyaline changes and the deposit of calcareous material in the stroma (*sarcoma petrificans*).

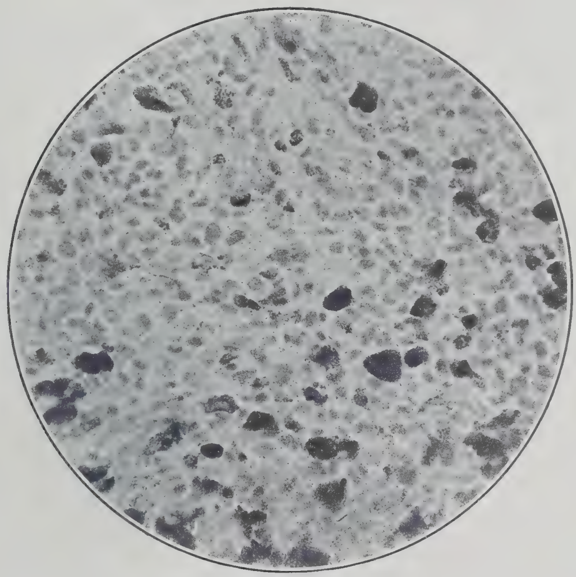

FIG. 106.—Melanotic Sarcoma. Winckel No. 6, without ocular. (From the author's collection.)

Sarcomatous Tumors of Mixed Type.—Under this caption we can conveniently discuss those forms of sarcomata which represent cellular variations of the simple benign tumors.

In the *fibro-sarcoma* the connective-tissue nature of the growth is quite evident. As in the fibroma there are cells of connective-tissue type together with a homogeneous and fibrillar intercellular substance. In the fibro-sarcoma the cells are more numerous, the nuclei plumper, and the fibrillar substance less in evidence. As may be imagined, it is not easy in many cases, from the histological appearance alone, to draw the line between the benign fibroma and the fibro-sarcoma. It is well known to surgeons that a tumor which has existed for a long time and has been regarded as an innocent fibroma may recur after removal, exhibiting a limited degree of malignancy (recurrent fibroma). The transition from the fibroma molle to the fibro-sarcoma is, in fact, almost imperceptible. In another form of fibro-

sarcoma the structure is not so uniform, but there are more or less dense fibrous septa enclosing spindle-shaped sarcoma cells.

The *myxo-sarcoma* resembles the myxoma, except that the round and stellate cells are more numerous, while the mucoid and fibrillar intercellular substance is relatively more scanty. The lipoma may also be transformed in part into myxoma and subsequently into *lipo-myxo-sarcoma.*

The *chondro-sarcoma* is a highly cellular tumor derived from the chondroma. It consists in the main of closely aggregated round, oval, or fusiform cells with relatively little cement substance. But the true nature of the growth can be made out by recognizing here and there islets of unaltered cartilage.

Like the lipoma, the chondroma not infrequently undergoes myxomatous and, later, sarcomatous change—*chondro-myxo-sarcoma.*

The *osteo-sarcoma* is a tumor consisting of bone, the medullary spaces of which contain, not marrow, but sarcoma cells.

The *osteoid sarcoma* is similar, bony plates and spicules being formed which, however, are not ossified. Not infrequently subsequent calcareous deposit will convert the osteoid sarcoma into the osteo-sarcoma. These tumors are usually found in connection with the periosteum. The formation of bone is probably due to the action of the periosteal osteoblasts, which are in some way stimulated into activity, and, possibly, are carried out into the substance of the growth.

The supporting stroma of an angioma may occasionally undergo sarcomatous transformation (*angioma sarcomatodes*).

Myo-sarcoma, a tumor homologous with the fibro-sarcoma, myxo-sarcoma, and chondro-sarcoma, composed of undifferentiated muscle cells, is theoretically possible, but little is known about its actual occurrence. The vast majority of tumors described as myo-sarcomata are either rhabdo-myomata or else myomata presenting secondary sarcomatous transformation of the intermuscular fibrous supporting substance. They are more properly termed *myoma sarcomatodes*. Some rare muscle tumors, forming metastases in the internal viscera, have been described in connection with the uterus, and are believed by those recording them to be composed of immature muscle cells. These would be the true *myo-sarcomata.*

II. TUMORS OF EPITHELIAL TYPE.

Under this heading we would include all tumors whose most notable feature is that they contain epithelial elements. There is invariably, however, more or less connective tissue present which serves as a supporting stroma or matrix, so that in a sense some at least of these growths may be regarded as being of *mixed* type. The proportion of connective tissue varies in different cases. In some instances the main mass of the growth is composed of connective tissue which is definitely proliferating, the epithelial structures simply keeping pace to form an external covering. Such tumors have more in common with the fibromata and myxomata than with the epithelial growths. In others, the epithelial

elements are in such excess that a highly cellular tumor is produced. A good example of the former is the papilloma; of the latter, adenoma and carcinoma may serve as examples. Strictly speaking, before we should regard a given tumor as of epithelial type, we must be certain that there is a primary autonomous new formation of epithelial structures. It is in many cases, of course, difficult to be sure of this. Where, for example, epithelial structures are present in small amount, it is not impossible that they may not be newly formed, but simply entangled in the course of the excessive proliferation of the fibrous stroma. Therefore, there are not a few tumors whose status is somewhat doubtful. They are, consequently, discussed here largely as a matter of convenience.

As we have seen above (p. 329), *papillomata* assume varying forms and are of diverse etiology. Many of them have affinities with the inflammatory hyperplasias rather than with the true neoplasms; others are fibromata, lipomata, and myxomata which have come into special relationship with epithelial structures. Still others are definitely the result of the proliferation of epithelial elements. The first two classes have been sufficiently dealt with already (pp. 303 *et seq.*), but the last-mentioned demands further attention. This subject will be more conveniently discussed, however, in connection with the adenomata and cystomata.

Adenomata.

Adenomata are tumors arising from glands or gland-like structures, the structure of which they more or less perfectly reproduce. When in the viscera, they form circumscribed, nodular masses usually encapsulated. On free surfaces they are apt to be compound, polypoid, villous, or papillomatous. As a rule they grow slowly and rarely attain a great size.

Adenomata are moderately common and are found more especially in the mamma, kidney, liver, suprarenal, thyroid, uterus, and the mucous membrane of the alimentary tract; occasionally, also, they originate in the sudoriparous, salivary, and lachrymal glands. I have been fortunate enough to find and report a unique adenoma of the pancreas arising from an island of Langerhans (*Journal of Medical Research*, November, 1902). Structurally, the adenomata consist of an epithelial part and a connective-tissue part. The epithelial cells usually resemble somewhat closely those of the gland from which they arise, and in a general way are arranged so as to reproduce the acini and ducts, but here the resemblance usually ends. The regular structure of the normal gland and the relative proportion of its parts are considerably departed from. The connective tissue forms a stroma supporting the glandular portion and in different cases varies greatly in amount.

It is usual to divide the adenomata into two classes, according to their histological appearance, namely, the *tubular adenomata* and the *alveolar adenomata*, to which may possibly be added a third, the *papilliferous adenomata*. In the first-mentioned variety the epithelial cells are in large measure arranged after

the fashion of tubules or ducts possessing definite lumina. The alveolar or acinous adenoma reminds one of the normal acini of the gland, save that they are much more numerous, and are apt to be larger and more highly convoluted. In the case of the papilliferous form, the connective tissue forming the walls of the acini or ducts proliferates actively and, pushing the epithelial cells before it, encroaches upon the cavities, which often become dilated, in the form of polypoid or papillary protuberances.

Adenomata are formed, probably, much in the same way as glandular structures are normally produced, namely, by the proliferation of the epithelium, which penetrates the connective-tissue stroma and assumes the form of acini and tubules. These structures are, however, produced in excess, and in some instances there is evidence that the stroma is not entirely passive, but participates in the overgrowth also.

Certain of the adenomata are of considerable practical importance to the surgeon. Of these may be mentioned the adenomata of the mamma, kidney, suprarenal, thyroid, prostate, testis, and uterus.

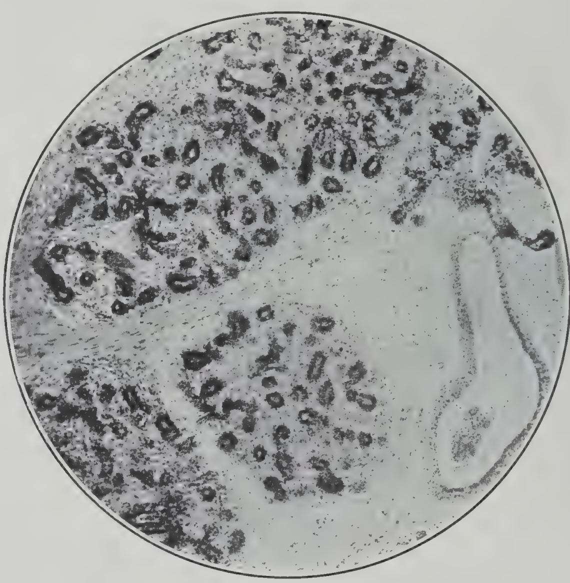

Fig. 107.—Fibro-adenoma of the Mamma, of the Acinous Type. Winckel No. 3, without ocular. (From the author's collection.)

The adenomata of the breast take the form of nodular masses, the size of a hazelnut or larger, which are movable, elastic, and moderately firm. On section they are lobulated, and the lumina of the dilated gland tubules and acini can be recognized on the cut surface. Occasionally, areas of softening and cystic degeneration can be detected. They are of slow growth, do not cause retraction of the nipple, do not involve the axillary nodes, and do not recur after removal.

Histologically, several varieties may be recognized. Certain of the fibromata of the breast, above described, contain a more or less notable amount of glandular

structure. This may in some cases be an accidental admixture, but not infrequently with the overgrowth of the fibrous tissue there is undoubted new formation of glandular acini. Such tumors are often termed *adeno-fibromata* and *fibro-adenomata,* according to the relative proportions of the two elements present (Fig. 107). Pure adenomata of the mamma are somewhat rare. In some instances the newly formed glandular tissue results in the production of new terminal acini, associated together in groups, and lined with cubical epithelium, resembling somewhat closely the structure of the normal functioning gland. In others the growth is erratic, presenting irregular tubules lined with cubical and cylindrical cells. The various tubules and acini are bounded externally by a doubly refractile basement membrane. The recognition of this structure is important, for so long as it remains intact the growth is benign. Should the glandular elements proliferate irregularly and appear outside the basement membrane the tumor would be called a carcinoma. Many carcinomata of the breast originate in a simple adenoma and are hence called adeno-carcinomata, though there is no doubt that some arise directly from the glandular epithelium.

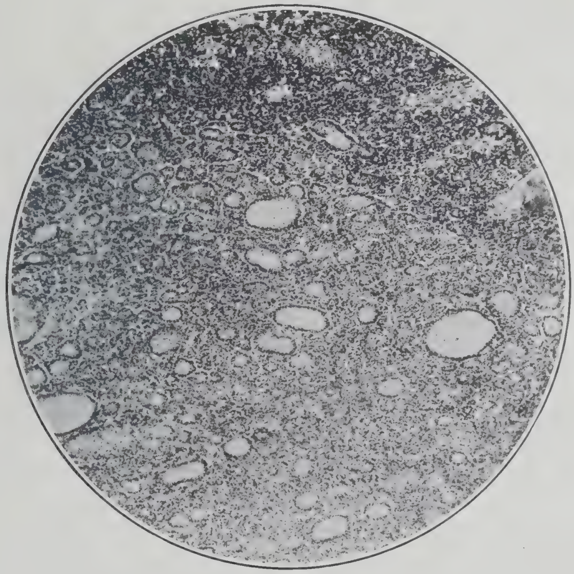

Fig. 108.—Fœtal Adenoma of the Thyroid. Winckel No. 3, without ocular. (From the author's collection.)

Adenomata of the kidney are well-defined, rather soft, growths, of whitish color, which may be microscopic in size or may attain that of a walnut. Histologically, they are tubular, acinous, or papilliferous. The epithelium resembles more or less closely that of the secreting tubules. Some of the renal adenomata, however, have been shown to originate in misplaced suprarenal "rests" (benign hypernephromata). The adenomata of the kidney may become malignant.

Glandular benign tumors of the thyroid are of three types, *simple, fœtal,* and

papilliferous adenomata. The simple adenoma consists of follicles filled with colloid, resembling those of the normal thyroid. The epithelium lining the follicles is cuboidal or somewhat flattened. In the supporting stroma can be seen here and there islets of similar cells, resembling embryonic thyroidal structure. The growth may be diffuse (colloid struma) or localized and encapsulated. In some cases the colloid increases in amount, the follicles enlarge, the intervening walls atrophy and rupture, and thus cysts filled with colloid result (cystic goitre). Simple adenomata are usually single, but may be multiple.

The fœtal adenomata resemble in structure the fœtal thyroid. The cells are arranged in solid columns and clusters, in which occasionally minute lumina may

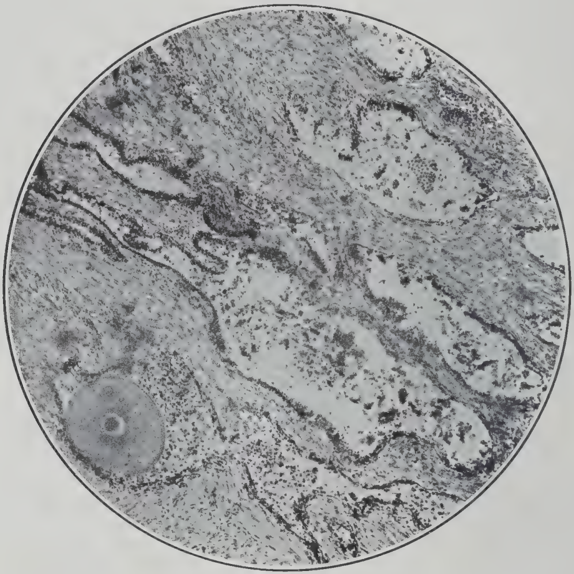

Fig. 109.—Adenomatous Enlargement of the Prostate. An "amyloid body" may be seen at the lower part of the section. Winckel No. 3, without ocular. (From the author's collection.)

be discovered, but colloid is not present. The tumor is usually encapsulated, is whitish or reddish in color, and of soft consistence. As a rule it is quite vascular. (Fig. 108.)

The papilliferous adenomata usually originate in the walls of old cysts, but very rarely they are true papilliferous adeno-cystomata, comparable to those of the ovary.

Adenomata of the suprarenal capsule are quite common, but rarely attain any size. It is interesting that misplaced suprarenal tissue may give rise to adenomata in unlikely situations such as the kidney, peritoneum, and broad ligament.

The enlargement of the prostate that so often occurs in old age is due to an overgrowth of the glandular portion, the fibro-muscular stroma, or both. When the glandular elements are increased the prostate is enlarged, spongy, and moderately soft, and on pressure a fluid rich in cells can be expressed. The overgrowth

is usually generalized, but nodular masses may be formed. Microscopically, the acini of the gland are increased in numbers, are enlarged, tortuous, and often dilated. They frequently contain concretions (Fig. 109). There issome doubt as to whether the condition should be regarded as a simple glandular hyperplasia or a true tumor formation. The importance of the condition lies in the fact that the overgrowth leads to encroachment upon the urethra, which it obstructs. This is particularly apt to be the case when the so-called middle lobe is enlarged. As a consequence the bladder becomes hypertrophied, later dilated, and at times inflamed (Fig. 94). Even the ureters, the pelvis of the kidneys, and the kidney proper may become dilated from the excessive pressure.

Adenoma of the testis originates in the seminiferous tubules and may be solid or cystic. When present, the cysts are filled with a clear mucoid material (*cystadenoma mucosum*) or with a cheesy detritus (*cystadenoma atheromatosum*). In the latter variety the cysts are lined with a thick, somewhat keratinized epithelium. Cartilage and muscle fibres are not infrequently present, suggesting the teratoid nature of the growth. In a few cases there may be a sarcomatous transformation of the stroma (*adeno-sarcoma* or *cystadeno-sarcoma testis*).

In determining the nature of a glandular overgrowth occurring in connection with the lining membrane of the uterus considerable difficulty is encountered. In the uterus the mucosa lies directly upon the muscular wall without the intervention of a submucosa. The uterine glands also occasionally penetrate into the muscle. In inflammation, as for example in endometritis proliferans, the tubules are enlarged, often dilated, and increased in numbers, sometimes forming intercommunicating spaces. The resemblance to a tumor is close. Moreover, round-celled infiltration which might in other cases be of diagnostic value is present in both. In making the differentiation between endometritis and adenoma on the one hand, and between adenoma and adeno-carcinoma on the other, regard must be had to the extent of the glandular proliferation. In many cases, however, we must be in doubt. Some authors describe an adenoma of the uterus consisting of numerous enlarged, dilated, and intercommunicating tubules held together by a somewhat scanty stroma. The tubules are lined by a single layer of irregular, compressed-looking cylindrical cells, often ciliated and showing mitoses. The tumor is apt to infiltrate and forms a connecting link with the adeno-carcinomata (*adenoma uteri malignum*).

From what has just been said on the subject of adenomata of the prostate and uterus, it may be inferred that there is a close resemblance between simple glandular hyperplasia and adenomatous new formations, and this is, indeed, the fact. Structurally, the appearances in both are practically identical, any difference being merely that of degree. If we take, for example, the mucous membrane of the stomach and intestines, it is not unusual to find, in the neighborhood of chronic inflammatory patches and especially ulcers, that the tissues are undergoing marked hyperplasia. This must be regarded as an attempt at regeneration, but

while in some cases it leads to the repair of the injury by the formation of normal mucous membrane, it not infrequently occurs in excess and produces tumor-like polypoid outgrowths. Histologically, such structures conform in appearance to tubular glands, but deviate somewhat from the normal in that the glandular elements are more irregularly disposed and branched. In a certain sense there is an atypical glandular formation. The overgrowth may also be so active that the glands are dilated and we get a kind of papillary excrescence. Such formations occur also in the absence of any pre-existing inflammation, and in these cases must be regarded as true hypertrophies. Here we begin to enter the region of the adenomata. Irritation of some kind would seem to be of some importance in the etiology of these growths. The fact that they are sometimes found at birth suggests in some cases the influence of developmental anomalies. An interesting confirmation of this is found in those adenomata which are traceable to misplaced portions of the suprarenals and thyroid gland. These can hardly be attributed to anything but the proliferation of cells or remnants of organs which have in the course of embryonic development been dislocated from their natural environment.

The adenomata are usually considered to be benign tumors. They may, however, take on atypical and aberrant growth, and may, therefore, pass on into carcinoma. There would appear to be a gradual transition of forms between the simple adenoma and the frankly malignant adeno-carcinoma. This, again, has led to some confusion of terms and ideas. There are certain tumors, that histologically must be classed with the simple adenomata, which on occasion are competent to produce distant metastases. The secondary growths in their turn present the structure of a plain adenoma. In a sense they are comparable to the chondroma, which acts occasionally in a manner similar. Such are some of the adenomata of the thyroid, the intestinal tract, the ovary, and the uterus. It is perhaps a matter of taste whether we term these, with certain authors, *adenoma malignum* or *carcinomatosum*, or *carcinoma adenomatosum.*

CYSTOMATA.

Closely allied on the one hand to the fibromata and on the other to the adenomata are certain forms of cystic growths known as *epithelial* or *proliferation cystomata.*

It would be well here to keep constantly in mind the distinction that exists between a *cyst* and a *cystoma.* In a broad way a cyst may be defined as a pathological cavity containing fluid or semifluid material. The term does not connote any new formation of tissue. As examples we may cite the degenerative cysts that often are met with in tumors and inflammatory infiltrations, the result of necrosis and liquefaction of the substance, retention cysts, the developmental cysts occurring in connection with the embryonic fissures, and parasitic cysts. A

cystoma is a true tumor, resulting from the proliferation of a matrix that tends to form cavities. It is possessed of powers of independent growth.

The true cystomata generally arise in structures that contain epithelium. When occurring elsewhere they must be derived from "cell rests" or misplaced embryonic tissue, and should, therefore, be classed with the teratomata (*q. v.*). The cavities in a cystoma are single, multiple, or multiloculated, and are lined by epithelial cells. The material contained within the cysts is fluid or semifluid, presumably the result of the secretory activity of the lining cells, or in part a transudation from the lymph- and blood-vessels. The fluid often, also, contains cholesterin, fatty and caseous matter, blood, pigment, and cell detritus.

With regard to the gross structure these tumors may be largely cysts or, again, they may be partly solid and partly cystic.

Histogenetically, they are found to have affinities with the adenomata and the papillomata.

Favorite sites for proliferation cysts are the thyroid, breast, ovary, kidney, liver, broad ligament, wall of the uterus, and vagina.

The first method by which cystic tumors may develop from the adenoma is well illustrated in the case of the thyroid gland. Here, in the lobules of the gland new acini containing colloid are formed, constituting an adenoma or colloid struma. The colloidal secretion gradually increases until the walls of the acini become greatly distended and finally rupture, many of them thus becoming confluent. In this way there are sometimes formed cystic cavities of considerable size containing colloid and often blood. This might be termed the *glandular type* of cystoma.

The second type is the *papilliferous cystoma.* A good example of this is to be found in the *intracanalicular papilloma of the mamma.* This originates in a fibroadenoma of the organ. Such tumors usually contain at some part or other somewhat dilated ducts and acini (tubular adenoma). The interstitial stroma of connective or mucoid tissue proliferates, forming papillary processes that project into the glandular spaces, gradually distending them. These outgrowths are covered with epithelium and may become highly complicated and exuberant in their career, in some cases even extending through the ducts of the nipple and appearing externally.

The ovarian cystomata are, too, of great practical importance, being among the most common neoplasms affecting these organs. They are unilateral or bilateral, unilocular or multilocular. They produce symptoms largely by their size and weight, but one variety, the papillary cystadenoma, exhibits a marked tendency to become malignant.

The most frequent variety is the *simple cystoma.* This is commonly unilateral and is composed of one cyst of relatively large size, together with several smaller subsidiary cysts. The cyst wall is tough, thin, and translucent, and the cavities are filled with a viscid, mucinous fluid, either clear and colorless or

mixed with cell detritus and blood. The cyst wall is composed of two layers of fibrous tissue, the outer dense, the inner vascular and more cellular. The lining membrane of the cavities is usually composed of a single layer of high cylindrical cells, but, in the larger cysts, of short columnar, cuboidal, or even flattened cells. The lining epithelium often extends outward into the wall of the cysts, forming simple or compound gland tubules. It is rare for the epithelium to be stratified. Occasionally the epithelium is ciliated.

A second, but rare form is a pedunculated multilocular cyst of moderate size, usually unilateral, lined with cylindrical epithelium. The contents of the cysts are thin, more serous than in the first form, light yellow or greenish in color, and rich in albumin.

The most important type, however, is the *papillary cystoma,* or *cystadenoma papilliferum.* This is a multilocular or, occasionally, unilocular cyst, which is apt to be bilateral. The cysts are usually smaller than those of the simple cystadenoma and are more or less filled with warty, villous, or tree-like formations of connective tissue covered with ciliated epithelium (Fig. 110). In a few cases cilia are absent or are present only on the papillæ. The growth extends between the layers of the broad ligament or forms a pedunculated mass springing from the surface of the ovary. The fluid contained within the cysts is thin, watery, often dark colored, and more serous than that of the simple cystadenoma. Not infrequently the cauliflower-like excrescences appear externally, either because the outer wall of certain of the cavities has given way or because of an actual invasion of the wall by the new growth.

This form of cystadenoma has a great tendency toward excessive and independent growth. According to Pfannenstiel about one-half the cases in time become malignant. In rare cases the tumor spreads along the peritoneum, forming local metastases that reproduce the cystic and adenomatous character of the primary tumor.

The pathogeny of ovarian cystadenomata is not at all clear. As Orth has pointed out, all sorts of transitional forms exist between the simple and the papillary varieties. This, together with the fact that in all varieties the cysts may be lined with ciliated epithelium, suggests that the ovarian cystadenomata have a common origin. This is by no means necessarily so, however, for it has been shown that under certain circumstances non-ciliated epithelium may acquire cilia. Theoretically, ovarian cystadenomata may arise from the epithelium of the follicles, from the superficial germinal epithelium, from certain tubules of the paroöphoron (Waldeyer), from displaced "rests" of the ciliated tubal epithelium (Kassmann), or from remains of the Wolffian body (Koelliker). The developmental origin of many of them is supported by several observations. Cystadenomata are usually met with during the period of sexual activity and, often, in both ovaries. Cases have been reported where sisters or mother and daughter have been affected, suggesting a hereditary peculiarity. Again, it is not uncommon to

find the combination of a cystadenoma and a dermoid. Authorities are not agreed whether to assign the same mode of origin to the simple cystadenomata and the papilliferous forms. Orth is inclined to attribute the majority of them to the same precursor, the germinal epithelium.

The cystomata of the kidney rarely attain a large size. The congenital cystic kidney is in some cases probably to be regarded as a true tumor or cystadenoma. Certain of the smaller ones may be traced to misplaced suprarenal "rests." The alveolar, tubular, and papillary adenomata of the kidney occasionally give rise to cystic growths not unlike those of the ovary in outward appearance.

Multiple cysts of congenital origin have been found in the liver. The condition is often associated with congenital cysts of the kidney. Little is known positively about them. They are probably to be regarded as true cystomata, com-

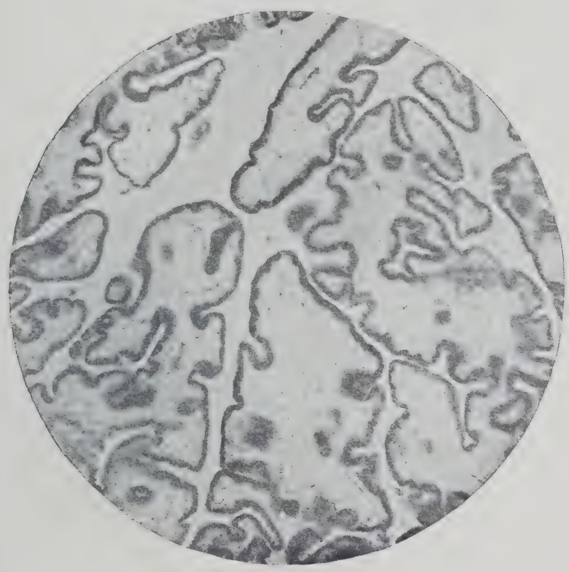

Fig. 110.—Papilliferous Cystoma of the Ovary. Leitz objective No. 3, without ocular. (From the author's collection.)

parable in most respects to those of the kidney. Some of them may possibly start from suprarenal "rests" in the liver, inasmuch as suprarenal tissue has occasionally been found there.

Carcinomata (Atypical Epi-, Meso-, and Hypo-lepidomata).

Just as we have malignant new growths that are cellular variations of connective tissue and are known as sarcomata, so we can recognize malignant neoplasms of epithelial origin—the carcinomata. We may pursue the parallel further. As we get sarcomatous transformation of tumors derived from fibrous tissue and its congeners, as for example in fibromata, myxomata, chondromata, osteomata, so we may have carcinomatous metamorphosis of the benign epithelial growths—the papilloma, adenoma, and cystoma.

A carcinoma may be defined as a malignant tumor arising from epithelium. It possesses a remarkable tendency to local infiltration, sooner or later undergoes partial necrosis, and commonly produces secondary new growths in distant parts (metastasis). Carcinomata arise wherever epithelium is found, from the superficial epithelium of the skin, from the epithelium lining the alimentary tract and lungs, and from the invaginations of epithelium constituting the secreting glands. Carcinomata occasionally, also, manifest themselves in structures where epithelium is not normally present, as, for instance, deep down in the neck (in connection with the branchial clefts) and in the walls of dermoid cysts. Such an occurrence does not invalidate the general rule, for in such cases the tumors originate from embryonic epithelium which has become displaced and separated from its proper environment in the course of development. From the standpoint of embryology the epithelium from which carcinomata are developed may belong to any of the three primitive cell aggregations—ectoderm, entoderm, or mesoblast.

Histologically, carcinomata resemble the epithelial or fibro-epithelial structures from which they spring, with one important difference to be referred to anon. We find masses of epithelial cells of varying size and shape enclosed in spaces or alveoli, and supported by a connective-tissue stroma carrying the blood-vessels for the support of the tissues. The connective tissue and the blood-vessels never penetrate the epithelial-cell masses.

We can best understand, perhaps, the nature of carcinoma and the sequence of events that give rise to it if we consider for a moment the normal proliferation of epithelial structures. Epithelial tissues are among the most active in the body. Owing to the demands of function and their exposed position they are subjected to a great amount of wear and tear. This is quickly made good. The reparative powers of epithelial cells, however, are not only sufficient for these lesser calls, but are competent to replace extensive losses of substance. Moreover, under certain circumstances, as, for example, under the influence of irritation, large masses of tissue, composed principally of epithelial elements, can be produced. In addition to the proliferation of the epithelial cells in such cases there is a new formation of connective tissue, which acts as a supporting stroma and carries the blood-vessels. Such a structure repeats somewhat closely the appearances of the original tissue from which it sprang and is, therefore, called *typical*. Of this nature are certain papillomatous and polypoid outgrowths, warts, and condylomata, before referred to, that stand in an intermediate position between the simple inflammatory hyperplasiæ and the true tumors. But we may go further than this. If we take, for instance, a chronic ulcer of the skin and subcutaneous tissues, it is not unusual to find marked evidences of proliferation of the epithelial cells of the cutis at the periphery of the lesion. The epithelium is thickened and tends to penetrate deeply into the loose connective tissue resulting from the inflammatory action. In fact, the ordinary histological features of the epithelioma or epidermal carcinoma are simulated with remarkable accuracy. Such a growth must, therefore, be

termed *atypical.* There is, however, this important fact to be noted: *the downward extension of the superficial epithelial cells only extends as far as the confines of the altered connective tissue, and ceases so soon as the source of irritation is removed.* In other words, the new growth is *not autonomous.* In the carcinomata, on the other hand, while the proliferating epithelial cells retain a somewhat close resemblance to those from which they are derived and with which they are in anatomical continuity, they grow wildly and without regard to the neighboring structures. The orderly arrangement of the original tissues from which they spring is departed from. Structurally speaking, there is no normal prototype of the carcinoma. This, then, is the crucial point in the differentiation of carcinomata from other forms of epithelial-cell proliferation: *the overgrowth is not only atypical but it is aberrant.* Inasmuch as the proliferation appears to be the result of forces inherent in the epithelial cells themselves, the carcinomata, like other tumors, are autonomous formations. To illustrate. Let us take the case of carcinomatous transformation of the adenoma of the breast, an occurrence that is by no means uncommon. In the adenoma we have a more or less abundant new formation of acini, ducts, and tubules, closely resembling those of the normal functioning gland, enclosed in an orderly fashion within a basement membrane. The glandular nature of the growth is quite evident. The epithelial cells of such an adenoma may at times take on excessive action. They proliferate more rapidly, are heaped up in places, and finally break the bounds of the limiting membrane and appear in the intervening fibrous stroma. Here they form rounded, oval, elongated, or irregular solid clusters, in which the arrangement into acini and ducts can no longer be traced. These masses are, however, in direct continuity with the epithelial elements of the original tumor. Such an atypical and disorderly growth of the epithelial cells constitutes a carcinoma. In a similar fashion papillomata, especially those of an adenomatous character, such as the papillomata of the bladder and rectum, and certain cystomata, may take on malignant action.

In the cases just cited, the carcinoma originates as a cellular variation of a tumor of organoid type. But this is not the only way. Carcinoma may originate directly from epithelium without going through an intermediate organoid stage. This occurs, for instance, in the skin and mucous surfaces. In the development of squamous-celled carcinoma or epithelioma of the skin, the part is first enlarged owing to the simple hyperplasia of the Malpighian layers, the follicles, and glands. But soon the aberrant character of the growth becomes evident. The proliferating cells begin to penetrate the subjacent fatty and connective tissue as finger-like processes and strands of cells, which in parts coalesce, forming a sort of network. The normal relationships of the various tissues entering into the part are quickly obliterated, as the growth becomes exuberant and erratic (Fig. 111). In a similar way, carcinomata of the mucous membranes, as of the stomach and bowels, begin with hyperplasia of the glandular elements, the cells of which increase in size and numbers, penetrate the basement membranes, burst through the muscu-

laris mucosæ, and eventually appear in the muscular wall. The term *adeno-carcinoma,* so often employed, refers to a carcinoma that reproduces the glandular type in a recognizable degree. The large majority of carcinomata are adeno-carcinomata.

Having considered the manner in which carcinomata originate, we may properly inquire into their mode of growth. This depends in large measure on the nature of the supporting stroma of the part involved. In the case of connective tissue we have a meshwork of fibres, between which lie the cells proper. The spaces form an intercommunicating system and are the radicles of the lymphatic channels. Now, if a mass of epithelial cells begins to proliferate, and does not extend to the surface after the manner of a typical growth, it extends downward and at once enters this system of tissue spaces, where it continues to grow.

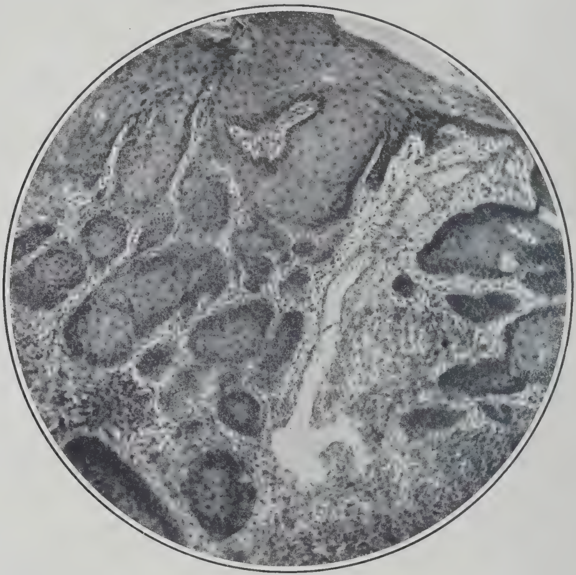

Fig. 111.—Epithelioma. This section shows very well the aberrant downward growth of the superficial epithelium of the skin. Winckel No. 3, without ocular. (From the author's collection.)

The epithelial-cell clusters invariably lie within the lymph spaces and extend by way of the lymph channels. In hardened sections, in which the epithelial cells have shrunk away from their boundaries, it is often possible to detect a layer of endothelium lining the alveolus, similar to that lining the lymphatics.

In a general way, the cells forming a carcinoma resemble those of the epithelial structures from which they arise. Close study, however, will reveal some notable differences. The carcinoma cells are often larger and possess larger nuclei; there is considerable variation in shape; and degenerative changes are often to be observed in the protoplasm. Single cells of relatively great size, containing a single large nucleus, can occasionally be seen; or, again, cells may be seen which contain a multitude of nuclei. The nuclei are rich in chromatin and stain deeply. The process of cell division is, moreover, abnormal. In place of simple division

of the nuclei, we get the most complicated and irregular nuclear figures. Some of the cells contain vacuoles filled with fat or hyalin and appear to be phagocytic, for they may enclose leucocytes, red corpuscles, or plasma cells. In fine, the differences taken together indicate an overplus of vegetative energy. The degenerations so commonly found are a natural accompaniment of this, the cells growing so fast that they cannot obtain sufficient nourishment.

The clumps of epithelial cells, lying in the alveolar spaces, present great variations in size and shape. As a rule, the newly formed epithelium forms a solid mass which ramifies in the connective tissue, not unlike the roots of a tree. At the periphery of the main mass, small isolated clusters of cells may often be found, having no visible connection with the rest.

The distinction between cells and stroma is usually well preserved, but if the stroma be loose and cellular it is hard to determine the limits of the new growth. In the case of loose connective tissue and fat the epithelial cells grow wildly in all directions, so that the alveolar arrangement is lost. We often find the carcinoma cells extending in long rows as a somewhat diffuse infiltration.

It should be noted that the carcinoma cells preserve, so far as may be, the physiological characters of the epithelium from which they are derived. If we take the case of the epithelioma of the skin, the finger-like processes that invade the deeper tissues are composed of cells that develop, grow old, and die, just as do those of the superficial epithelium. We find, for example, that the cells at the periphery of the cell masses correspond with those of the Malpighian layer. As we proceed toward the centre the cells gradually become flattened and are converted into keratohyalin. This gives rise to curious translucent bodies having a concentric lamination resembling the layers of a pearl or onion. These are the "epithelial pearls" or "cell nests" that are so conspicuous a feature of the epitheliomata of the skin (Fig. 112). The same tendency is manifested in the columnar-celled carcinoma of the rectum. The proliferating epithelial cells come to be arranged side by side, their long axes pointing in the same general direction. As a result we get the columnar cells grouping themselves about a central lumen, thus reproducing more or less faithfully the original tubules and acini. The regularity of this formation is often, however, lost in consequence of the exuberant growth, so that groups of cells become forced into the cavity and there form acini, solid masses, and complicated loops. Pressure, too, of the rapidly growing cells will naturally modify the arrangement. Carcinomata of the thyroid also give rise to secondary growths that assume the alveolar structure of the normal gland and may even produce colloid. This tendency to retain the original characteristics of the parent cells is, as one would expect, most marked in the case of slowly growing tumors, while it is lost in the more exuberant growths. It may, moreover, be present in one part of a tumor and absent in another.

Mention has been made above of degenerative disturbances which are not infrequently present in the specific cells of carcinomata. These take the form of

simple coagulation, of colliquative necrosis, or, again, of colloidal and hyaline transformation. Necrosis is apt, of course, to occur in rapidly growing tumors, where the vascular mechanism is unable to keep pace with the epithelial proliferation. Necrosis usually occurs in the centre of the cell masses, or, in other words, at the point most remote from the nutrient blood-vessels.

From the histologist's point of view the appearance of the cells constituting a carcinoma forms a ready means of classification. Thus we may recognize a *squamous-celled carcinoma* (carcinoma plano-cellulare), a *round-celled carcinoma* (carcinoma globo-cellulare), and a *cylindrical-celled carcinoma* (carcinoma cylindro-cellulare). It should be remarked, however, that while the carcinoma cells tend to reproduce the characters of the epithelial cells from which they spring, yet they do not always perpetuate these. The more rapidly growing the tumor and

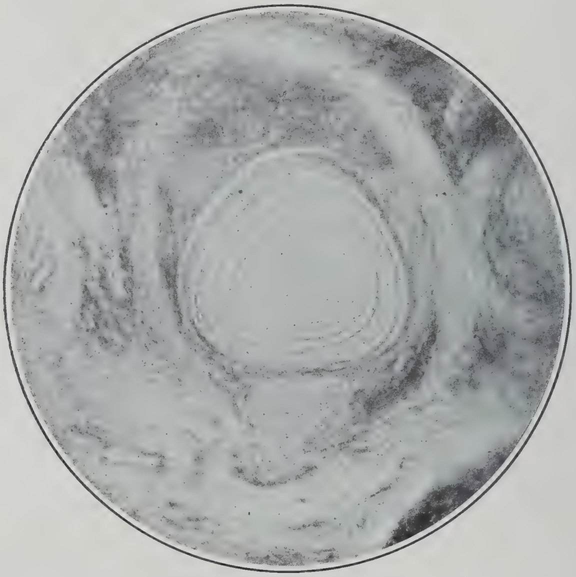

Fig. 112.—Epithelial Pearl or "Cell-Nest," from an Epithelioma of the Lip. Winckel No. 6, without ocular. (From the author's collection.)

the farther removed its cells from their original progenitors, the more widely do the specific carcinoma cells deviate from the type. Thus, a carcinoma of the cylindrical-cell type may in parts consist of clusters and off-shoots of round cells. This has been by some termed metaplasia of epithelium. It is more likely, however, that the round cells are merely young and immature forms of cylindrical cells. This is often well illustrated in the case of metastases which may be quite unlike the parent growth.

The *squamous-celled carcinoma* (epithelioma) may arise in any part of the body where stratified pavement epithelium is found. It occurs, therefore, in the skin, especially that of the face and lip, in the buccal mucous membrane, the tongue, œsophagus, anus, vulva, vagina, vaginal portion of the cervix uteri, penis, and

conjunctiva. Rarely, epitheliomata may arise from papillary warts and nævi, from atheroma cysts, and from dermoids. Very exceptionally, a squamous-celled carcinoma may originate from parts that contain no squamous cells. This is known to occur in the uterus. Von Rosthorn and Zeller have described a metaplasia of the columnar cells lining the uterine cavity into pavement cells, and from these a squamous-celled epithelioma may develop.

A squamous-celled epithelioma results from the invasion of tissues and organs by proliferating epithelial cells derived from stratified pavement epithelium constituting a protecting membrane or lining a cavity.

Histologically, it consists of a supporting stroma usually of connective-tissue or muscle, or both, in which are alveoli filled with cells of epithelial type. The cell clusters usually appear to be distinct and isolated, but serial sections show that they are united at various levels by lateral processes, so that the epithelial masses have really a plexiform arrangement. In the epitheliomata of the skin, the cells at the periphery of the alveoli are round, cubical, or short columnar, and are placed at angles to the surface of the stroma, in this resembling the germinal cells of the rete Malpighii. Many of them can be recognized as "prickle" cells. As we approach the centre, the cells become more flattened and spindle-shaped, and gradually lose their nuclei. The central cells, as before mentioned, retaining their physiological peculiarities, are gradually transformed into an almost structureless keratohyalin material. The concentrically arranged cell masses may calcify at the centre, liquefy, or swell up, or may become converted into colloid. They are not always found in epitheliomata, or, if they are, they are present in very small numbers.

Carcinomata of this type are particularly apt to break down on the surface, thus forming an ulcer, the edges of which are soft and swollen.

A special form of epithelioma of the skin, that demands a word or two, is the so-called "rodent ulcer." This begins as a small ulcer of the skin, not infrequently on the face near the eyelids. It spreads irregularly at the periphery, while the older parts cicatrize and heal, again becoming covered with epithelium. The growth is essentially chronic and may last many years. Histologically, it is a superficial epithelioma of the skin, but must be regarded as the least malignant form of this type of cancer.

The *round-celled carcinoma* is composed of spaces filled with round cells or cells which have been rendered polyhedral from pressure. Since the various diameters of the cells are approximately equal they have been termed *isodiametric*.

Round-celled carcinomata arise in glands, like the mamma, salivary glands, and liver, and in glandular tumors, that contain isodiametric epithelium. Occasionally, they may arise from the cylindrical epithelium of mucous surfaces and glands, the cells of which have been transformed into the isodiametric type.

Histologically, we may recognize a *large alveolar round-celled form*, in which the spaces contain a large number of isodiametric cells closely packed together,

and a *small alveolar round-celled variety,* in which a smaller number of cells, usually from two to ten, are to be found.

The *cylindrical-celled carcinoma* originates in mucous membranes, glands, ducts, and tubules provided with cylindrical epithelium. We find it, therefore, very commonly in the stomach and intestines. In a typical case, the alveoli consist of cylindrical cells arranged so as to enclose a central cavity, in this suggesting the normal structure of the gland. Certain of the cells in question are goblet cells. The lumina generally contain fluid, mucin, and disintegrated cells. The cells lining the alveoli may form a single layer, or, again, they may be stratified. Here and there the smaller alveoli can be seen to contain masses of round cells, which are solid buds of young growing cells springing from the cylindrical cells lining the spaces. These may be so numerous as to constitute the tumor a transition form between the cylindrical-celled and the round-celled carcinoma.

The shape of the epithelial cells closely resembles that of the cells of the normal mucous membrane, and it is curious how faithfully the glandular appearance of the new growth is preserved. A reference to Fig. 113 will show how closely such a carcinoma may resemble the simple adenoma. Certain features, however, will aid us in making the differential diagnosis. Thus, cylindrical-celled carcinomata usually ulcerate early, much earlier than do the adenomata. The important clinching point is, however, the presence of epithelial cell masses in parts where normally epithelium is not present. In the adenoma, say of the intestinal mucous membrane, cell masses and alveoli are produced which closely resemble the growing processes of the carcinoma. There is this important difference, however, the proliferation of the cylindrical cells in the adenoma is entirely confined to the mucosa; in other words, it lies above the muscularis mucosæ. In the cylindrical-celled carcinoma, on the other hand, the cells soon break these bonds, pass into the submucosa, and eventually invade the muscular layers and the neighboring structures. Goblet cells, which are so important a feature of the gastro-intestinal mucous membrane, are fairly numerous in the adenoma, while they are much rarer in the carcinoma.

Having discussed the nature and appearance of the epithelial cells that constitute a carcinoma we pass on to consider the character of the supporting stroma. This also presents considerable variations. While it forms part of the tumor mass and grows with it, it cannot be said to be an integral part of the tumor. The stroma of the tumor represents in part the normal tissues of the locality that has been invaded by the epithelial cells. Thus, in an epithelioma of the skin the stroma consists of the subcutaneous connective tissue and fat together with sebaceous and sudoriparous glands. As the tumor grows there is undoubtedly a new formation of the interstitial connective tissue advancing *pari passu* with it. This may possibly be interpreted as an attempt to form a vascular tissue competent to carry nutriment to the growing epithelial structures. Possibly, too, it is to some

extent a reaction on the part of the stroma resulting from the irritation produced by the presence of cells foreign to the normal tissues.

The stroma usually consists of fibrous connective tissue, though in exceptional cases it may be composed of muscle, as in carcinoma of the uterus, or of bone, as in secondary carcinoma of bone. Here and there in the stroma can be seen isolated clumps or, sometimes, a diffuse infiltration of round cells, resembling the lymphoid cells of the lymph nodes and the lymphocytes of the blood. Plasma cells are present also, but are somewhat less numerous. With this there are evidences of proliferation of the connective-tissue cells proper, but this is usually in the background. Occasionally the proliferation is so marked that the interstitial stroma comes to resemble a sarcoma. Occasionally giant cells can

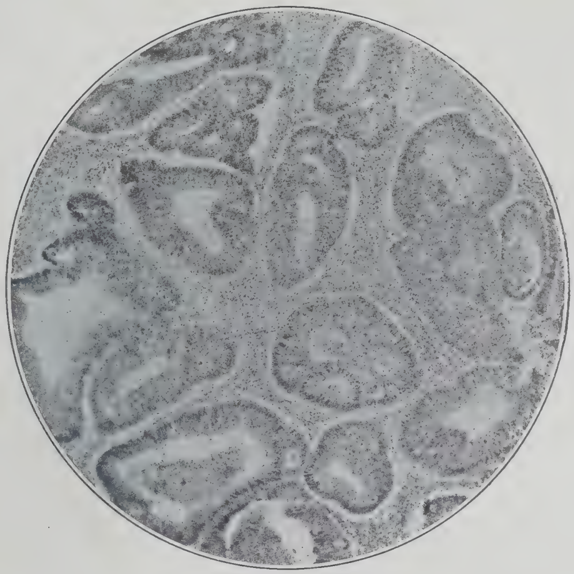

Fig. 113.—Columnar-celled Adeno-carcinoma of the Rectum. Winckel No. 3, without ocular. (From the author's collection.)

be seen in the stroma, similar to those sometimes found in the neighborhood of foreign bodies.

The relative amounts of epithelial-cell masses and of stroma vary greatly in different tumors and even in different parts of the same tumor. This forms a convenient basis on which to divide carcinomata according to their gross appearances. If the fibrous connective tissue greatly predominate we speak of a *scirrhous carcinoma.* In such cases the epithelial-cell clusters are small, often attenuated, and atrophic-looking. If the epithelial cells be numerous and arranged in small clusters bounded by a delicate connective-tissue wall, we have an *alveolar carcinoma.* If the stroma be scarcely apparent so that we get a soft brain-like growth, we call it a *medullary* or *encephaloid carcinoma.* A carcinoma in which stroma and epithelial elements are about equally divided is termed a *simple carcinoma.*

The margin of a carcinoma is rarely sharp. The greatest growth is at the periphery, and the tumor extends in lines into the adjacent tissues. There is never any attempt at the formation of a capsule. In the neighborhood of the growth can often be seen small foci of epithelial cells either separated from the main mass or attached to it by a delicate thread of tissue. The invasion and destruction of the healthy tissues in the immediate vicinity of the growth are a marked feature. The destruction of the tissues seems to be brought

Fig. 114.—Carcinoma of the Lesser Curvature of the Stomach, with Ulceration. (Pathological Museum of McGill University.)

about, not so much by pressure or by phagocytic action of the carcinoma cells, as by simple lack of nutrition, all the available pabulum being appropriated by the tumor.

Secondary Changes in Carcinomata.—I have above mentioned the fact that degenerative phenomena are commonly to be found in carcinoma cells. When the growth is of any size these become quite marked. Thus, in the centre of the cell clusters, we get fatty degeneration, vacuolation, atrophy, and even necrosis. In this way large portions of the alveolar contents are destroyed. The nuclei disintegrate, the cytoplasm fragments, and we get a dirty-looking granular

detritus that stains badly. When the necrosis is superficial it leads to ulceration (Fig. 114). In internal carcinomata, as for example those of the liver, the detritus is in part absorbed, and the nodules formed by new growth soften and become depressed in the centre, or "umbilicated" as it is called.

Certain of the degenerative changes are so striking that they stamp the tumor as something out of the ordinary. An instance of this is the *colloid* or *gelatinous carcinoma*, found most often in the alimentary tract and mamma, less often in the ovary. It forms a nodular growth or a diffuse infiltration. On section the tumor shows in some part or other, or possibly throughout, a characteristic translucent, gluey, or gelatinous appearance. This is due to a mucinous or gelatinous degeneration of the epithelial-cell clusters. The carcinoma cells may in time entirely disappear, and the spaces are then filled with a homogeneous, glassy

Fig. 115.—Colloid Carcinoma. Winckel No. 3, without ocular. The carcinoma cells are greatly degenerated and have been replaced by colloid, which can be recognized as long stringy fibrils. (From the author's collection.)

substance that under the microscope appears as structureless fibrils striking a purple color with hæmatoxylin (Fig. 115). In other cases the fibrous stroma undergoes myxomatous transformation—*carcinoma myxomatodes,*—either alone or with mucinous transformation of the epithelial cells as well. Thus the whole growth may become translucent and gelatinous.

A rarer form of carcinoma is that in which hyaline transformation of certain of the epithelial cells or of the stroma takes place—*carcinoma cylindromatosum.* It occurs in the skin, the intestine, and in glands.

Pigmented carcinomata—*melano-carcinomata*—have been described, but are still rarer. The pigment lies partly in the epithelial cells and partly in the stroma, giving the tumor a gray, brownish, or black color.

Methods of Extension and Metastasis.—If we examine a growing carcinoma, we find that it is sending out at the periphery processes of epithelial cells into the tissue spaces, spaces that, as we have already seen, are to be regarded as the ultimate radicles of the lymph channels. This is termed *extension by infiltration* (Fig. 116). Some few carcinomata are almost as sharply defined at the margins as a benign growth, but in most there is undoubted infiltration of the surrounding soft parts, and in some this may be quite far-reaching. Sometimes, also, we find small nodules at some little distance from the periphery, similar in appearance to, but quite distinct from, the primary growth. These are the result of minute emboli of carcinoma cells within the lymphatic channels leading from the part. This is known as *extension by dissemination.*

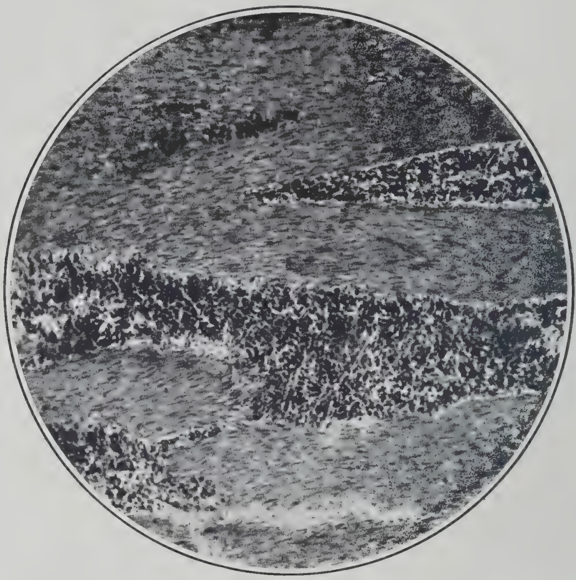

Fig. 116.—Carcinoma of the Stomach. This specimen shows very clearly the infiltration of the muscular wall with epithelial cells. (From the author's collection.)

Small clusters of epithelial cells may also break away from the main mass of the growth and be carried by the lymphatics or, occasionally, by the blood stream, to distant parts, where they set up independent foci of disease. This phenomenon is called *metastasis.* In general the first manifestation of metastasis occurs in the regional lymph nodes nearest the primary growth (Fig. 117). If we examine one of these nodes in the early stage of the process, we find small foci of epithelial cells at the periphery of the node in close relationship to the afferent lymphatic channels and sinuses. At first, one sees the lymphoid cells between the epithelial-cell masses, but soon they atrophy and their place is taken by connective tissue. The metastases in general resemble the primary tumor, except that they are not so apt to retain the functional peculiarities of the cells from which they are ultimately derived. Thus, in metastases from an epithelioma of the skin we do not so often get the formation of the epithelial "pearls," and in adeno-carcinomata

the glandular appearance of the original growth is not so completely preserved. When the regional lymph nodes are thoroughly infiltrated, the masses of carcinoma cells pass out by the efferent lymphatics and invade the system of nodes next in order, or cancerous emboli may pass through the first series of nodes without involving them, and attack those more remote.

Metastasis by the blood stream is rather uncommon in the case of the carcinomata, though it is the rule with the sarcomata. Carcinomata of the stomach and intestines, however, commonly spread to the liver through the portal system, (Fig). 118, and carcinomata of vascular regions, like the penis, may extend through the blood sinuses and vessels. The new growth may directly invade the vessels destroying the wall and appearing within the lumen, or may reach the

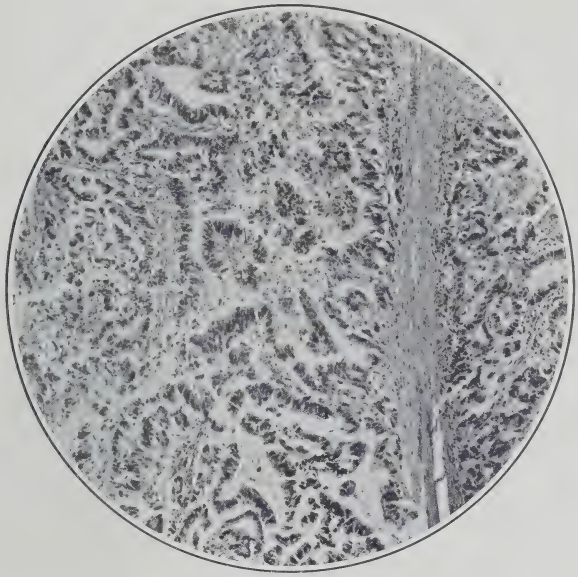

Fig. 117.—Secondary Invasion of a Lymph Node with Columnar-Celled Carcinoma. Winckel No. 3, without ocular. (From the author's collection.)

blood through the lymph-vascular system. Generally speaking, emboli from carcinomata of the gastro-intestinal tract reach the liver, those from tumors situated elsewhere reach the lungs. Exceptionally, invasion may take place in a direction opposite to the course of the lymph stream—*retrograde embolism.*

Extension of a carcinoma may also take place by *implantation.* In carcinoma of the kidney, secondary tumors may arise along the ureter and in the bladder. In carcinoma of the ovary, secondary nodules may appear in the Fallopian tubes and in the peritoneum. In the intestine, small secondary growths may be found in the mucosa below the original mass. In all these cases the dissemination of the growth appears in large part to be determined by gravity.

Carcinoma in its extension always takes the line of least resistance, and we find it spreading along the tissue interstices, and along the perineural and perivascular lymphatics.

III. THE TERATOID TUMORS.

In the foregoing pages we have had under discussion tumors that are members of the great family commonly known as the *Blastomata*. It remains for us to consider the second main group, the *Teratomata*.

The blastomata have been dealt with at considerable length, comprising as they do the vast majority of tumors commonly met with. The teratomata, being much rarer, are of not so much practical importance to the surgeon, though they are of the greatest importance in regard to the question of tumor formation. We will, therefore, in this place consider them only in a sketchy way.

A teratoma is a tumor the characteristic feature of which is that it is composed of cells or tissues that normally do not occur in the affected part, or at least are not present at the period of bodily development at which the growth mani-

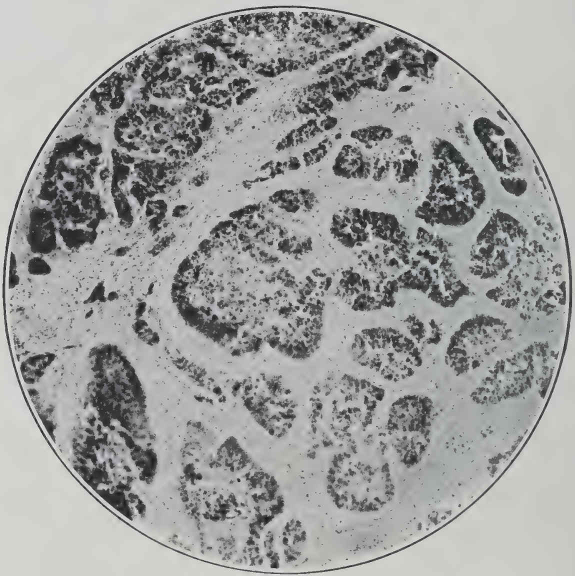

FIG. 118.—Secondary Carcinoma of the Liver. Winckel No. 3, without ocular. (From the author's collection.)

fests itself. The simplest form of teratoma is represented by a single tissue or a cyst (*simple teratoid tumor* or *cyst*), but as a rule more than one tissue and more than one germ layer are represented (*mixed tumor*). The term "teratoma" is often applied in a narrower sense to the more complex growths, while tumors consisting of derivatives of all three primitive cell-layers are called *embryoid tumors* or *embryomata*. The tissues entering into the composition of teratomata arise either from the *Anlage* of the affected individual (*monogerminal, endogenous,* or *autochthonous teratomata*), or from those of a second individual (*bigerminal ectogenous teratomata; fœtus in fœtu*).

Occasionally, sarcomatous or carcinomatous transformation may occur in the tissues of a teratoma, constituting a *malignant teratoma*.

As all teratomata are due to proliferation of misplaced or redundant cells, it is evident that we may meet with all degrees of complexity, from the simplest epidermoid or implantation cyst to the most complicated malformation and monstrosity. For information on the latter phase of the subject the reader is referred to works on teratology.

Warthin gives the following classification of teratomata, which is as simple as any:

Teratomata:
- 1. Simple teratoid tumors.
- 2. Simple teratoid cysts.
  - Ectodermal.
    - Dermoid.
    - Epidermoid.
  - Mesodermal.
  - Entodermal.
- 3. Complex teratomata and teratoid cysts (embryoid tumors and embryomata).
- 4. Malignant teratomata.

Simple teratoid tumors consist of a single variety of tissue or at most of only a few forms of tissue. Tumors belonging to this group are the hypernephromata, rhabdo-myomata, chondroma of the mamma, salivary glands, skin, testis, etc.; adeno-myoma of the uterus and broad ligament; leiomyoma of the kidney; osteoma of muscles, skin, mamma, tongue; lipoma of the meninges; coccygeal and lumbo-sacral lipomata and myo-lipomata. Most of the tumors of this group are to be regarded as heterotopic tumors, arising from autochthonous fœtal "Anlage," but some possibly may be bigerminal inclusions.

Simple Teratoid Cysts.—Ectodermal teratoid cysts include cysts lined with stratified squamous epithelium, without other skin structures (*epidermoid cysts*), and cysts whose wall contains hairs, glands, and fat (*dermoid cysts*), in this resembling skin. Epidermoids are sometimes due to injury, as in the so-called implantation dermoids. I have met with one such case where the penetration of the palm of the hand with a blunt piece of wood was followed by the formation of a small cyst of epidermoid character. One of the most interesting forms of epidermoid is the *cholesteatoma,* found in the meninges, the hypophysis cerebri, and the middle ear, among other places. It is a spherical or nodular tumor, varying in size from that of a pea to that of an orange, and on section has a glistening, waxy appearance. Histologically, it is composed of flattened, scale-like cells, devoid of nuclei, arranged in a laminated fashion. The central portion tends to degenerate and is often filled with a pultaceous mass containing plates of cholesterin.

Mesodermal and entodermal cysts originate in misplaced entodermal and mesodermal "Anlage," or the persistence of fœtal ducts and glands. They are lined with columnar epithelium, sometimes ciliated, and are found most frequently in the female genital tract, less often in the peritoneal cavity, intestine, close to the trachea and bronchi, and in the lungs, pleura, tongue, neck, liver, and kidneys.

Complex teratoid tumors and cysts are found in the same situations as the forms above described but are commonly met with in the sexual glands and about the coccyx. They consist of a great variety of cells and tissues, squamous and columnar epithelium, ciliated epithelium, skin, nerve, fat, striped and unstriped muscle, cartilage, bone, and glands. The tissue represented may be adult or immature. The ovarian dermoid may be taken as a type.

This is a thick-walled cyst filled with a fatty, pultaceous substance, lanolin, and sometimes wisps of hair. At one point of the inner wall is a prominence covered with hairs and occasionally containing teeth. This may contain masses of bone, suggesting a jaw. The prominence referred to consists of all the structures of the skin. The cyst is lined in places with ciliated epithelium. In the cyst wall derivatives of all the three primitive germinal layers may be found.

Malignant Teratomata.—Any of the above-mentioned tumors and cysts may undergo secondary malignant transformation. Some behave as malignant from the first. The more complicated solid growths, especially those of the genital tract and mediastinum, are those most apt to exhibit this tendency.

A word or two should be said about the *chorio-epithelioma malignum,* sometimes called *deciduoma malignum.* Inasmuch as this tumor is derived from the cells of one individual proliferating within the tissues of another, it can properly be included with the teratomata.

Chorio-epithelioma malignum is a new growth originating in the fœtal epiblast of the chorionic villi. It grows rapidly, infiltrates, and forms metastases. The growth is polypoid or fungous, projecting into the cavity of the uterus, is of reddish color, and of soft, friable texture. Microscopically, the tumor resembles a carcinoma or sarcoma, or both, but there may be in addition syncytial or plasmodial masses, or even villi. The growth originates in the proliferation of the syncytium and the Langhans' layer of the chorionic villi. The syncytium is thickened and the cells of the Langhans' layer tend to grow toward the surface. The deeper parts present an alveolar arrangement. The resulting tumor has no stroma and no blood-vessels. Hemorrhage into the growth and necrosis are common features.

THE RESULTS OF TUMOR FORMATION.

All tumors produce effects by their size and weight. The neighboring structures are pressed upon and as a result undergo atrophy, or, if movable, they may be dislocated. Pressure upon blood-vessels leads to obstruction of the circulation, œdema, thrombosis, embolism, or necrosis. Pressure upon nerves causes pain and may lead to paralyses. Pressure upon the ducts of glands may result in retention of secretion and dilatation of the organ. Tumors on the extremities may interfere with locomotion and the free action of joints. Pedunculated growths, especially when of large size, are apt to undergo necrosis and ulceration, owing to the interference with nutrition that eventually takes place.

Secondary infection may result in local inflammation and even generalized septic manifestations.

Malignant tumors, in addition to the conditions mentioned above, which are largely the result of mechanical forces, and, therefore, are particularly well exemplified in the case of the benign growths, possess the power of infiltrating and destroying the structures in which they may be growing. Their power of metastasis has already been referred to. When superficial, carcinomata may ulcerate and become inflamed. The malignant growths also give rise to a peculiar form of generalized marasmus, known as *cancerous cachexia.* This is manifested by great weakness, wasting of substance, and an earthy color of the skin. It owes its origin in part to the pain and discomfort caused by the growth, and in part to the interference with the functions of the body, notably digestion; it may also in some measure be attributed to septic absorption; and, finally, it should to some extent be considered a systemic manifestation of poisonous substances emanating from the new growth.

THEORIES OF TUMOR FORMATION.

By THEODORE A. McGRAW, M.D., LL.D., Detroit, Michigan.

True tumors or neoplasms have been aptly defined as "new growths of tissue which have no physiological connection with the body." The essential features of this definition have been generally accepted, but there is hardly a pathologist of note who has not tried to improve it by variations in the mode of statement or by explanatory additions. These efforts have generally ended in failure, for the reason that it is impossible to define exactly and minutely conditions which we do not understand. We speak of the physiological connection of normal tissues with the organism of which they form part, because we have become assured, from observing certain constant phenomena, of the existence of physiological laws, which are violated in the growth of every neoplasm, but our knowledge is so vague and indefinite, and the mechanisms by which vital processes are carried on are so beyond all human understanding, that we cannot formulate them in terms which convey exact ideas. We cannot, however, understand the abnormal without having first obtained more or less clear conceptions of the normal; and it is necessary, therefore, on entering upon the study of tumors, to inquire into the nature of the law which is violated in their growth, even though we may not hope to account for its existence or explain the method on which it acts. We may do this, perhaps, to best advantage by considering briefly certain facts in embryonal and post-embryonal life.

Every animal organism begins life in the impregnated egg. From this cellular unit spring an enormous number of cells, whose generation takes place with a predestined order. In millions of individuals of the same species there comes almost precisely the same sequence of changes, from which there is, only in rare cases, any deviation whatever. The original cell divides by a process of segmentation into a cluster of cells, which soon proceed to arrange themselves in layers, assum etheir proper relations to each other, become differentiated, and eventually develop into various tissues and organs under a compulsion the nature of which is absolutely mysterious and inscrutable.

On studying these manifestations of vital energy, we soon come to see that every animal organism becomes such by virtue of its own inherent force. In itself lies the power which compels every cell within its limits to expend its energies only in such ways as will contribute to the general good.

The environment has an influence on the development and growth of the cell, for it affects those external conditions of protection, temperature, nutrition, etc.,

on which every living thing is dependent; but it has no power to initiate the evolution of the embryo nor to keep the proliferating cells in proper control.

To a complex organism, however, the existence of a governing power within itself is a primal necessity, for if its constituent units should multiply without regard to its necessities, if it could neither limit their propagation nor get rid of them when they had become useless, it would necessarily die from its own weakness. Accordingly we find in every normal animal body evidences of the existence of just such a controlling force, and, if we study carefully the changes which occur in the growing embryo and in the nutritive processes of the adult, we may distinguish two modes of action by means of which it produces the necessary results. By the one it forms continually new cells and tissues and sometimes new organs, while it removes by the other all débris, destroys all cells, tissues, and organs which have accomplished their end and become effete, and causes the disintegration and absorption of all living matter which has become useless or obstructive. There is nothing more wonderful in nature than the working of this unseen and unobtrusive force. We see in the embryo masses of cells form themselves into organs, which perform some obscure function and then disappear. The Wolffian body has for the most part diminished to nothing at the close of the sixteenth week of gestation, but one portion, that destined to form the sexual organs, has increased in size and importance. In the formation of the vagina we see the same processes of tissue building and tissue destruction going on simultaneously, the Mueller's ducts coalescing in the middle line and the central cells disintegrating and disappearing. If we watch the growth of bones, we find taking place together a growth of bone and a destruction of cartilage. Everywhere we see the exercise of a controlling power which compels all cellular action to proceed on certain defined lines.

We may say, in a certain sense, that Nature abhors a cellular anarchy. Sometimes she is betrayed in the processes of evolution into an excess of energy, and more germinal material is formed than can be utilized; but she will then try to regain her normal standpoint by the destruction and removal of the superfluous mass. A notable example of this tendency may frequently be seen in cases where extra fingers and toes are found on newborn children. In many cases the useless members are well formed, but so located as to be of no use to the organism. The effort to remove them may be noted in the absorption of the tissue which connects them to the extremity, for in the majority of instances they hang to the hand or foot by a mere thread of skin. We see similar evidences of a governing power in adult life, where there is constant loss of organic units which must be met by a corresponding regeneration. The useless and effete cells are destroyed and new ones appear to perform their functions. Large organs even may be formed to replace defects produced by disease or injury, as when, after removal of the thyroid, the subsidiary thyroids which exist in some persons develop into large and active glands, or as when a kidney doubles in size

to compensate for the loss of its neighbor. The processes by which these results are obtained are never obtrusive in normal conditions, and may often be best studied when the vital operations are accentuated by disease or when in wounds they are exposed to the eye. In the latter case we may see manifest evidences of the twofold action: First, proliferation of cells; and, second, their eventual destruction or disappearance; and, in addition to these, the exercise of an inhibiting force which limits a cellular proliferation, after it has reached its proper limit.

In a deep wound we may see granulations forming with great rapidity, but with this enormous cellular growth there goes a contraction of the new tissue, which lessens the area of the wound and draws its walls together. Finally, when the granulations have reached the surface they cease to multiply and become passive, and give place to a new kind of cellular activity, that of the adjacent epidermis, which then grows over the wound surface and gives it its protecting mantle. This accomplished, the new tissue gradually changes into a hard, dense scar, with the disappearance of the cells whose activity produced the healing. Thiersch imagined that this subsidence of connective-tissue formation on reaching the level of the skin was due to a power residing in the local tissues, which enables them to repel the encroachment of cells of a different kind on their domains. This idea has been modified in various ways, especially by German authors. All of them recognize local influences which limit the germination and growth of cells by opposing to the cellular activity the active or passive resistance of a living environment. The resistance may take the form of pressure, or of secretions unfavorable to cellular growth, or of monopoly of nutriment. Other authorities, extending this idea until it embraces the entire organism, imagine that the organic solidarity is due solely to a balance maintained between antagonistic tissues.

It seems to me more reasonable to believe that behind all the phenomena of generation, growth, and nutrition there exists in every complex organism some unconscious intelligence which directs and controls the vital processes. I cannot conceive how any balance could long exist between constantly changing tissues and organs which may lose their powers of resistance by any chance disease or injury, unless there is some regulating force inherent in the organism as a whole. It is only by means of such a controlling power that that perfect co-operation and co-ordination of the cells of an organism can be maintained which are the very essence of physiological unity. Upon the perfection of this controlling force depends the perfection of the individual. It is when this force is weakened or lost that we see groups of cells develop into those useless and destructive masses which we call tumors.

When we call in review these facts of organic life, the question inevitably arises whether any tumor, even the most innocent, can be regarded as a mere local affair. If the power of control is normal, no tumor can grow; if lost over any portion of the body, this loss may indicate a vital defect.

Before the days of Virchow, the word "constitutional" was used to designate certain diseases which were supposed to arise from morbid conditions of the blood. Since the advent of cellular pathology and the demonstration that all maladies originate in perverted cellular action, the word has lost all meaning to the pathologist.

As regards neoplasms, the expression used by W. Roger Williams, of Bristol, England, "that nobody nowadays thinks of wasting his time in discussing the obsolete riddle as to whether these diseases are of local or constitutional origin," represents, doubtless, the present attitude of the professional mind; and yet, this much may be said on the other side, that no riddle is obsolete that is unsolved. Pathological societies may put such questions on the shelf as unworthy of attention, but they will, nevertheless, reappear for discussion until the human mind has found a satisfactory solution. We may not say that tumors or cancers are constitutional in the old sense of that word, but when we are confronted, again and again, with certain phenomena for which we cannot account, we are compelled to ask ourselves whether the local manifestations represent the whole morbid action, and whether preceding that local affection and accompanying it, there may not be some unknown quantity of far greater importance. We have to ask ourselves, then, what is the nature of the force which co-ordinates all normal cellular activity, and how it is that it becomes paralyzed and inert. The growth of a tumor may indicate either that a single group of cells have become emancipated from the general control, or, on the other hand, that the power of the organism as a whole to govern its constituent units has become impaired. In the first case, the tumor may be of only local significance; in the second, we have a condition that involves the whole body in a common danger. The appearance, then, of even the most innocent neoplasm may have in it something portentous.

There are reasons for believing that this constitutional defect acts much more frequently as a cause of tumors than is generally believed. There are, first of all, the numerous cases of heredity, where neoplasms of various kinds appear in a family through several generations. There is no other way of accounting for these cases except on the theory that such families labor under defects of development and growth. Then, again, there are those cases where many and diverse tumors appear on the person of the same individual—cases difficult to understand on the theory of local origin. The fact that in old age, when the vital forces are weakened, tumors become common, points also to some general cause for their occurrence.

The most unanswerable argument for such a belief, however, may be found in the study of the metastases of malignant tumors. A melanotic sarcoma makes its appearance in some locality, and thence infects the whole system by sending its cells or their nuclei through the blood-vessels to all parts of the body. The cells lodge and multiply, and a secondary tumor is evolved, precisely like the

primary growth in structure. No tissue in the person so affected can withstand the invasion, and in the course of a few months the patient dies.

In this history we see two violations of organic law. The first is the original growth of useless cells in the organism. This, however, might be accounted for on the theory of local severance of that group of cells from their physiological connection. The second is the repeated and successful implantations of these morbid cells in spots all over the body. The normal organism would resist the growth of such intruders and destroy them. This, in fact, is what occurs in artificial implantations of such growths in healthy animals; the graft either dies at once or undergoes speedy degeneration and disappears. It is only occasionally that an animal can be found which is susceptible to the inoculation of a true tumor, even from one of its own species.

While auto-inoculation of such growths is the rule, the successful implantation of such cells, in individuals other than the patient, almost never occurs. Surgeons and medical students may bury their hands in such neoplasms, carry away fragments under their finger-nails, and rub the pulpy mass into cuts and crevices of the skin, without ever showing the slightest symptom of the disease.

If we reason at all about the pathology of malignant tumors, we have no other choice than to assume that from the very beginning of such a disease there is a loss of control which involves the whole organism. Whether we should apply the word "constitutional" to such a weakness or defect is another question.

Cohnheim's Theory.

The most brilliant hypothesis regarding the origin of tumors ever advanced is that of the German pathologist Cohnheim. Like all other new ideas, however, this theory was an almost inevitable consequence of certain positive advances in knowledge, which enabled the student to look upon his subject from a novel standpoint.

With the advance of embryological and histological science, the theory had become generally accepted that the three blastodermic layers represented permanent divisions of tissues. It was believed that the ectodermal and entodermal layers would give rise only to cells of an epidermal or epithelial type, and that the mesodermal layer would produce only cells with peculiar characteristics of connective tissues. It was, however, a continual struggle to reconcile this theory with the fact that epithelial structures are frequently found embedded among the muscles and fascia in the form of dermoid cysts, that cartilage is found in tumors of the parotid gland, mammary glandular tumors in the axilla, etc. The question continually arose, whether, under the stimulus of morbid conditions, there might not occur a metaplasia of cellular elements which would entirely change their character.

The study of these conditions led to a possible solution of the problem, by the hypothesis of displaced or wandering germs. It was suggested that during the

period of embryonic development, in the many changes in the relations of tissues and organs, cells might occasionally become pushed out of their proper place and remain attached to other structures in abnormal positions and environments. If we assume that such cells survive their uncongenial surroundings, overcome the resistance of neighboring structures, and multiply and grow into masses of tissue, we have a plausible explanation of heterologous tumors. The enchondroma of the parotid appears, then, as a growth from cells which have been detached, in the formative stage of the embryo, from the germinal substance of the ear and have become attached to the parotid; the adenoma or mammary glandular tumor of the axilla springs evidently from detached portions of the nascent mammary gland; the dermoid cysts of the neck have originated from ectodermic cells which have accidentally been turned into the depths during the coalescence of the branchial arches. In this last instance a corroboration of the theory has been obtained from the history of those cases of dermoid cysts in the fingers of sewing-women, which are caused by implantations of minute portions of the epidermis by needle punctures. This theory of displaced germinal matter is so plausible and explains so many otherwise inexplicable pathological conditions that it has met with general acceptance.

Cohnheim, however, evolved from this class of facts a theory covering the etiology of all neoplasms. He assumed that in most healthy animals more germinal matter is formed during the evolution of the embryo than can possibly be used for purposes of development, and that these superfluous cells might persist indefinitely in the organism long after the period had passed when they could enter into physiological relations with the rest of the body. He supposed that such redundant germinal matter might date from any period of embryonic life, from the earliest period after impregnation to the full completion of development, retaining in its latent condition the same capacity for multiplication as that possessed by embryonal material in the same stage of organization.

Reasoning by analogy from the normal to the abnormal, he instanced the life history of the uterus. This organ, when impregnated, begins to grow, forming new muscular tissue, until it measures, after the expulsion of the fœtus, four or five times its original volume. It then undergoes the process of involution, when the superfluous uterine tissue disappears, leaving the organ slightly larger than the virgin uterus. This sequence of events occurs in every successive pregnancy, the growth after impregnation being followed by the destruction of the new tissue after childbirth. Cohnheim urges that there is only one explanation of these events possible. There must exist, in the uterus, germinal matter which responds to the stimulus of pregnancy and then develops into adult tissue. In every pregnancy some of this store of embryonic material is used up, and finally the supply, after repeated pregnancies, becomes exhausted. In case pregnancy should not occur, these germs, responding to some other stimulus, may develop

abnormally into those fibro-muscular tumors so common in old virgins and in barren women.

Like the supposititious germs of the uterus, so, too, the hypothetical superfluous germs, left stranded in the tissues after the wave of evolution had passed by, might, under favorable circumstances, develop later in life into tumors.

If they were pushed out of the vital current at an early stage of development, before the cellular masses had become differentiated into tissues, they would retain that enormous generative energy which is common to that stage of life. Thence would originate those terrible cellular growths which we class together under the name "sarcoma," or, if residual from the ectoderm or entoderm, the various forms of cancer. The aggressive powers of such neoplasms are due, according to Cohnheim, to the unexpended embryonic energy bottled up in such residual cells.

On the other hand, those tumors which originate from germinal matter at a later period, when this force has abated, after the tissues have become differentiated and fixed, would partake of a histoid character and grow more slowly and be less infectious.

As regards the development of such germs into tumors, two factors are necessary: one is the destruction of the resisting power of the normal tissues in which the germs are embedded, which would otherwise inhibit the growth of the abnormal cells; and the other is the application of some stimulus which would arouse the latent germinative energy.

While this theory may be accepted with some reserve in explanation of those congenital heterologous tumors due to the persistence and growth of displaced germinal matter, there are reasons for doubting its applicability to neoplasms of a different character. In view of the fact that one of the positive laws, in a complex animal organism, is that which determines the degeneration and ultimate destruction of all tissues which have neither present nor potential utility, it is difficult to believe that embryonic germs, subject like all cellular units to the action of this law, could persist for years together in a hostile environment, neither disappearing nor enlarging until the time came for them to grow into tumors. It seems more probable that adult cells may, under pathological conditions, change their nature and undergo a degeneration marked by great activity in the production of a low grade of offspring. Borst raises the question whether persistent dislocated germs are in reality more disposed to the heteroplastic development than others, and answers the question in the negative. Many of the examples he quotes, however, do not seem to me altogether pertinent. Accessory thyroids and suprarenal capsules, even though out of normal position, might, if able to perform the functions of those glands, have a perfect physiological connection with the organism. As regards accessory thyroids, there can, in fact, be little doubt that they have in some cases of loss of thyroid saved the patients from myxœdema. Indeed, it has been stated on good authority that

patients have even been cured of cachexia strumipriva by the successful implantation of thyroid tissue in the abdomen.

Borst is, however, undoubtedly right when he asserts that neither the aberration nor the persistence of supernumerary germs suffices in itself to produce neoplastic growths. There must, in addition, be added an unknown element which has thus far escaped all analysis.

We may say, however, of this theory of Cohnheim's that it is the only one advanced since the days of humoral pathology which has even attempted to account in a comprehensive and rational manner for the occurrence of neoplasms. It has acted as a wonderful stimulus to the whole profession in their studies of this dark subject, and, if it has not solved the whole riddle, it has, nevertheless, thrown great light on many of the questions nvolved. In the opinion of this great pathologist the cells from which tumors arise are from the very beginning abnormal.

It is interesting to note that the best men of to-day are adopting generally this point of view. They differ, however, from Cohnheim in this, that they do not regard embryonal aberration and persistence as the only abnormal conditions which are capable of producing the result. There is reason to think that under certain unknown conditions adult cells may undergo metaplasia, or, if you please, an anaplasia—a process of degeneration in which they so far simulate embryonic tissue that they acquire the power of rapid multiplication, although, unlike embryonal material, they are incapable of developing differentiated cells of a high grade. The cell progeny, even in histoid tumors, is badly formed and abnormal, while in the more malignant growths it represents the lowest form of undifferentiated cells. It always is marked by an absence of purpose and a uselessness, which are the most certain criteria of a true tumor. Ribbert derives the beginning of a tumor from the disruption of the physiological relations of a cell or group of cells to the organic whole. He believes that, whatever unknown forces may cause the disruption, when a cellular unit—either in embryonic or in post-uterine life—undergoes this change, it thenceforward leads a life of its own, regardless of all organic laws. He denies to these elements the power of producing in neighboring cells a similar metamorphosis, but asserts positively that they grow into tumors from their own inherent wild energy, by the multiplication of their own cells, pushing tissues aside and compressing them, or forcing their way into every crevice, and, in the malignant varieties, destroying the component cells.

In this respect Ribbert differs from some other pathologists, who ascribe to the original morbid elements a quality which enables them to cause in their sister-cells, by mere contact, similar morbid tendencies. In their view the tumor, originating in some great disturbance in cellular relations, grows by constant accretions from without, as one cell after another yields to the morbid impulse.

Max Borst, who has considered the subject exhaustively from all standpoints, concludes that the causes of neoplasms must be sought in the internal conditions

of the tissues in which they originate. He assumes the existence, in every case, of some congenital pathological quality of cells or tissues as the foundation for the formation of a neoplasm. Among the anatomical conditions for consideration in this connection he mentions, first, gross disturbances in the development of a region or organ or system; second, displacements of embryonal germs or disruption of such germs from their physiological connections without displacement; third, formations of superfluous germs in fœtal life; fourth, abnormal persistence of tissues which, in normal course, should have disappeared; and, fifth, failures in the differentiation of cells and minute disturbances in their idioplastic development.

Cheyne, of Edinburgh, assuming that every cell carries within itself a male and a female element, fancies that a disturbance of their relations may account for the formation of tumors, the female element becoming ungovernable and developing without physiological purpose. This theory lacks that foundation on established facts which alone can give a theory recognition by scientists. In the most of these theories the main element in the production of the abnormal growth is sought for in the aggressive action of the tumor cells, but some authors are disposed to lay the principal stress on the loss of resisting power in neighboring tissues. Thus Thiersch regards the atrophy of the connective tissues in old age as the primary cause of epithelioma, the epithelium growing wildly and irregularly because it is no longer checked by the conservative resistance of the tissue underneath. Regarding the organism as held together by a mutual balance and, to some extent, by antagonism between the various structures and organs, he conceives the loss of that balance to be of primary importance in the causation of all irregular cellular proliferation.

In all of these theories we may recognize the perception on the part of pathologists that there is something monstrous and portentous in the useless and aimless growth of cells which we class together under the name of true tumors or neoplasms. They occupy a peculiar and unique place in biology, for in no other class of vital processes can we witness the component units of an organism separating themselves from the organic whole and fastening themselves upon it as parasites and enemies.

The criterions on which we base our diagnoses of true tumors are, then, the evidences which we see of an organic rupture which endangers the very life and being of the animal. There can be no other symptom which indicates such a profound disturbance of a complex organism as the wild generation of useless cellular masses. In this common feature lies the mystery of all tumors, innocent and malignant. The essential nature of a lipoma or fibroma or osteoma is closely allied to that of a sarcoma or cancer. The same law is broken in the growth of one as in that of the other, and whether the result is a comparative innocuousness or a virulent malignancy would seem to be a matter of degree in morbid action rather than one of kind.

Before proceeding to the consideration of those theories which seek to explain the phenomena presented by tumors and cancers by the action of microscopic parasites, it may be well to turn our attention for a moment to the pathology of that most virulent class of neoplasms which are termed *par excellence* malignant. If we study the natural history of tumors with exclusive attention to the two ends of a long series, we may divide them into two classes, innocent and malignant, the first of which—although its members may cause damage by pressure or weight or mechanical interference with the blood supply or nerve conduction—is not in itself dangerous to life, while the second is primarily and always virulent and destructive. If, however, we take a more general view of the subject, and, instead of occupying ourselves solely with the extremes of the series, examine all with reference to their action upon the human body, we become convinced that the innocency or malignity of a tumor is not that which, biologically speaking, is its most marked characteristic. We shall find that, while the quality of virulence is much more marked in some tumors than in others, there is hardly any kind of neoplasm which may be said never to show it in some degree. There are many tumors, too, considered quite innocent, which have a tendency in time to change their characteristics in this respect and become malignant, either by a metaplasia of tissue or by offering a favorable soil for the growth of other neoplasms. Malignancy in a tumor is but another name for a tendency to make metastases. The cells, multiplying rapidly, cling no longer to their original habitat. Some of them push their way in long lines which may be traced, in epitheliomas, from the surface through crevices in the underlying tissues far into the depths; others, getting into the lymphatic spaces, are carried through the lymphatic vessels into the neighboring lymphatic nodes. Thence they reach, in time, the deeper lymphatics, and finally are discharged into the blood current. Others, again, as in sarcomas, involve the capillaries and veins at an early stage of the disorders, fill up the lumen of these vessels, and are sooner or later carried away as malignant emboli to lodge and grow in some distant part of the body. Where such tumors grow into the intestines or urinary passages, detached fragments may pass down with the excreta and become implanted lower down in the canals. When projecting into the serous cavities, they fall to the lower levels and cause numerous secondary growths.

Virchow long since pointed out the conditions which favored metastasis. They are, first, a tissue formation which permits the easy detachment of cells. In histoid tumors, like lipomas, fibromas, etc., the cells have become differentiated, multiply slowly, and have firm connections with the intercellular substance. Such cells are torn with difficulty away from their attachments, and metastases of such tumors are, of necessity, rare, and occur only when through some metaplastic change they have become more cellular and when the bonds of the cells to the stroma have become less rigid. It is evident that the drier and firmer tissues are, the more permanent they are in form. For this reason the

tendency to metastasis increases with the succulency of the tissues. The greater the amount of fluid contained in a tumor, the less stable are its component elements. The size and shape of the tumor cell also have an influence upon its tendency to metastasis. A small cell can more readily pass through a narrow channel than a larger one, and a round or spindle-shaped cell than one that is irregular or angular. More potent, however, than all of these qualifications for malignant growth is the possession of a great proliferating energy. Neither loose connections, small size, nor round shape could endow a fat cell with the aggressive force which enables a cellular unit to supplant and destroy its normal neighbors. There must be an inherent energy such as we see manifested in normal conditions only by embryonal cells. Whether malignant growths spring from persistent embryonic germs, as Cohnheim affirms, or whether there is a reversion of adult cells to an embryonal form and condition, as some assert, or whether, finally, without undergoing that kind of retrograde change, certain adult cells, when their physiological bonds are broken, may acquire the power of generating great quantities of a low-grade progeny, are questions which in the present state of biological science cannot be decided.

As regards the secondary tumors, it must be remarked that they invariably are composed of the same type of cells as that of the primary growth. The secondary tumors of a squamous-celled epithelioma are composed of squamous cells, those of a columnar-celled cancer have columnar cells, and the metastatic growths of a sarcoma invariably show their mesoblastic origin in the character of their cellular elements.

While adhering to the same type of structure as that exhibited by the primary growth, the metastatic tumors may, nevertheless, deviate from it in some particulars. The secondary and tertiary tumors will sometimes be more succulent than the primary and hasten more rapidly the disintegration, but, while the cells of such tumors undergo a metaplasia to a lower grade of development, they do not lose their distinctive qualities.

The course followed by malignant growths in their dissemination throughout the system is so similar to that pursued in infections of various kinds in which the active agent is a microscopic parasite that many pathologists have come to regard them as diseases of allied nature. Long before the days of cellular pathology, men were disposed to look upon cancers as parasites on the human body, and, while the grosser conceptions of a hundred years ago have been discarded, the belief has lingered in the human imagination. Of late years this view, modified to meet the present conditions of science, has been pressed upon the profession by many enthusiastic advocates who never tire in citing the various points in which cancer and sarcoma resemble the infectious granulomas.

In all these maladies the disease has a local origin; in all it spreads through the body by the same channels; in all it generates new foci in continuous and

contiguous tissues; in all the active agents which carry the disease to distant parts of the body are carried to their destination in the lymph and blood currents.

San Felice, Roncali, Plimmer, and other scientists in Europe, and in this country Gaylord, have found in microscopical sections of cancerous tumors peculiar figures which seemed to them to be different from all cells found in the normal tissues of the human body, and also from any form of cell degeneration known to the pathologist. They affirm that they have been able to grow these so-called Plimmer's bodies in cultures and to have caused fatal growths in the lower animals which have been inoculated with the germs. Plimmer regards these bodies as probably saccharomycetes, and is confident that they are the parasitic organisms which are responsible for the occurrence of cancerous tumors. These views, however, have not met with general acceptance. There is one point especially which distinguishes malignant growths of all kinds from those tumefactions which are caused by microbic infection. In the infectious granuloma the organism which conveys the infection is always and invariably the pathogenic microbe. The human tissue which nature builds around the focus of infection to wall it in, and if possible to destroy the infectious germs, is a granulation tissue. It is not a true tumor, but a false tumor, and the newly formed tissue is built up by the organism in its own interests as a defence against the invasion. Of widely different character is the pathology of a true tumor. In the human subject it springs from human cells which in some mysterious way have lost their physiological connections. From the multiplication of these cells arise the primary and all of the secondary tumors. If metastases are formed, it is because cells or their nuclei, which have been separated from the original growth, have floated away in the lymph or blood channels, have lodged elsewhere in the body, and have generated here and there a numerous progeny which compose new tumors. This new tissue is not formed in the interests of the organism, to protect it, but is itself the invader. It always shows in the character of its cells a likeness to the cells from which it is derived. When we study these conditions with reference to a possible parasitism as the causal factor, we have to ask ourselves whether it is possible for any parasite, animal or vegetable, so to act upon the human tissues as to break up their physiological bonds and cause their cellular units to grow into tumors, and, wandering from their primary seat, to establish colonies of the same kind in various parts of the body.

The burden of proof of a proposition of this kind, which is opposed to all of our experience, rests with those who advance the theory; but as yet it has not, in one single instance, been demonstrated. There are other considerations, too, which apply with almost equal force against this hypothesis. There is a relationship between tumors of all kinds which cannot be ignored. Malignancy is not confined to cancer or sarcoma, but is an attribute of many and various kinds of tumors. In our studies we may not limit our researches to cancer and our finds to Plimmer's bodies, but we must study the problem of malignancy wherever we

find it. If it is the result of parasitic infection, we have to ask whether this peculiar power over animal cells is limited to one kind of parasite or is common to many; whether the same protozoa or blastomycetæ which produce cancer also cause the phenomena of adenoma, malignant enchondroma, sarcoma, and other neoplasms with malign tendencies. It is evident that, if this were so, every malignant tumor would be a mixed tumor; for the parasites which attacked the epithelium would inevitably come in contact with other tissues, and, as the disease progressed, with all kinds of tissue. The lodgment of the parasite in the mammary ducts, for example, would first affect the epithelium and connective tissues of the breast; after that, as the disease progressed, the underlying muscles, and then the cartilages and bones, all of the various cells of these tissues being stimulated to a malignant and prolific generation of their own kind of cellular units. It is evident that this hypothesis could not bear criticism.

On the other hand, there are difficulties in assuming the existence of a large variety of parasitic forms each of which is gifted with the power of causing a wild cell proliferation in some particular tissue for which it has a predilection. As the matter stands to-day, the theory of a causal parasitic infection in the etiology of tumors has not been sustained. The results of inoculations with Plimmer's bodies have not been convincing, and all other phenomena which are relied on to sustain the hypothesis can be better explained on other grounds.

One of the most original of the theories in support of the doctrine of parasitism is that advanced by Kelling, of Dresden. It occurred to this author that the existence of cancer might be explained by the invasion of the human body by embryonal cells derived from other than human sources, and that these cells, more or less altered in character by their environment, might be the progenitors of the cancer cells. Having a foreign origin, they could not enter into physiological relations with the organism, but would multiply, grow, and destroy after the manner of parasites. He fancied that such cells might get into the human tissues from various sources, as, for instance, from embryonal tissues of pigs, lambs, fish, snails, etc.; but that the most frequent cause of trouble was due to the ingestion of raw impregnated hens' eggs which are used so commonly as food, the living cells obtaining entrance into the human tissues through some crevice or some ulcer in the alimentary canal. He examined many carcinomas from human subjects biochemically, in order to determine the nature of the contained albumin, and obtained reactions which indicated the presence of the albumin peculiar to fowls in about one-third of the cases. He chose for these investigations patients with gastric cancer secondary to gastric ulcer, who had, on account of the last-named disease, been fed with raw eggs. As a further test, he injected embryonal material from animals into animals of a different species, and produced thereby tumors which eventually caused death. He regards these tumefactions as true tumors of a malignant type. He further advances the opinion that the profession may hope, from the development of these biochemi-

cal examinations, to obtain a new and trustworthy method of diagnosticating cancer.

His ingenious theory and the conclusions which he draws from his experiments have not been considered by pathological experts as warranted by the facts. Like all hypotheses which would explain the origin of tumors on a basis of parasitism, this of Kelling meets with an insurmountable obstacle, in the histogenetic relations of all tumors and cancers. The cells of all tumors and their metastatic progeny are of the same type as the tissues from which the primary growth took its origin. The cells in human cancers could never, as Ribbert asserts, have arisen from the cells of a hen. Ribbert, whose pre-eminence in histological work no one can gainsay, denies positively the cancerous nature of the growths produced by Kelling's experiments, and regards them as enlargements due to irritation. From the biochemical side Kelling has been equally unfortunate, the investigation which Fuld conducted, with the purpose of obtaining the reactions of hen albumin from human carcinomas, having yielded only negative results.

Among the theories relating to the origin of tumors there remains yet to be mentioned one which is not new and which has not met with many advocates. The theory that tumors might be due to nervous disturbances was broached by Schroeder van der Kolk in the middle of the last century. There seemed at that time few facts to support it. Since then the study of acromegaly, in which disease enlargement of the bones has been associated with disease of the pituitary body, has given warrant for the hypothesis that there may be nervous centres whose function it is to regulate growths.

Recklinghausen has shown that the spots of pigment which are often in numbers on the body, appear at the ends of nerves and are associated with very minute fibromas. Not infrequently cancers and sarcomas develop from these spots. It would seem that the relations of the nervous system to tumors of all kinds might deserve a thorough and minute investigation. Should it ever be demonstrated that cellular generation and growth depend upon some as yet unknown nervous centre, the inference that the occurrence of tumors might be due to defects or diseases of that centre would not be unreasonable. It is not impossible that even before any nervous system comes into being the development of the embryo may be regulated by certain governing cells. If such hypotheses seem like idle speculations, it must be remembered that the solution of the problem, if it ever takes place, must come through the careful and patient investigation of every suggestion which may have in it a possibility of success.

There are many questions which arise with reference to neoplasms, which admit of answer only on a basis of accurate statistics. This phase of the tumor question has come recently into prominence through the intense interest excited by the statements of certain authors to the effect that cancer is greatly on the increase. The importance of establishing the truth or error of this opinion can

be hardly overestimated, and it is well, therefore, to ask whether there are any statistics, covering three or four decades, sufficiently trustworthy to warrant such a positive expression. The reliability of medical statistics depends upon the competency of the great mass of physicians who make the diagnoses and furnish the death certificates. It is evident that, as in every profession there are great numbers of badly educated, stupid, and indifferent men, there never can be any medical statistics which are absolutely exact. The utmost that can be hoped for, under the most favorable circumstances, is the attainment of results which are approximately correct. This consideration should, of itself, inspire caution in accepting any very positive statements based upon statistical reports. When, however, as in the case of the relative prevalence of cancer in different decades, the statistics have been compiled under constantly varying conditions, the profession should subject such statements to the severest criticism.

There has never been a period in the history of medicine when such radical changes have been effected as in the last forty years. During that period of time the profession has abandoned the old humoral theories of disease and has subscribed to the doctrines of cellular pathology. In surgery Lister's discoveries have caused a revolution in ideas and practice which can be described only as tremendous. Preventive medicine has become a science in itself, and health boards have been established all over the civilized world as permanent additions to the social organizations. In fact, the whole situation has so altered that the perusal of a medical book written before 1860 is like reading a work of Hippocrates or Galen. The profession, responding to the stimulus of the new ideas, has grown in stature. Its members have never before been so generally enthusiastic, and have never in the same lapse of time made advances in so many directions. The changes have taken place so rapidly that no two decades show precisely the same point of view on any medical subject. The doctrines relative to malignant diseases especially have been revolutionized, and the pessimism of the old humoral pathology has given place to hopes based on the theory of local origin, and the bias of the profession toward these diseases has become reversed. The general practitioner who formerly admitted with reluctance the existence of cancer when it was beyond hope is now disposed to regard every obscure malady as possibly malignant and to recommend operative procedures as a cure. The laity, participating in the confidence of the profession, are even too eager to seek in surgery a remedy for every ill. The operations on the abdomen have disclosed new pathological conditions and made clear what was dubious and uncertain. It was inevitable that these changes should become reflected in the health reports, and that the general advance in intelligence, in insight, and in efficiency should manifest itself in more accurate diagnoses. These changes in the pathological conceptions and in the methods of diagnosis and treatment, so widely adopted by the profession, are of themselves sufficient to account for an apparent increase in cancer, as shown by the health reports of a few cities; but, in

addition, we have to recognize the existence of other factors which also influence the character of statistical reports. One of these is the greater efficiency of the health boards of later over those of the earlier years. This is especially noticeable in some of the German reports. It is not many years since that the records of deaths in most civilized countries were kept in the most slovenly and careless manner. Even now the methods in vogue in many places are not beyond improvement. In Wuertemberg, for instance, according to Weinberg and Gaspar, in 1899, thirty-eight per cent of all deaths had occurred without the attendance of qualified practitioners. Of the deaths in Stuttgart, in 1879, thirty per cent of the deaths of men and twenty-seven per cent of those of women had taken place without competent medical attendance, but in 1901 this percentage had diminished to sixteen per cent of men and fifteen per cent of women.

Another element to be considered in estimating the character of the death certificates of the various decades is the increasing tendency of patients suffering from surgical maladies to seek relief in the hospitals; and still another is noticed by German authorities in the greater relative number of physicians at the present time—a change which has insured to the poorer patients more thorough examinations and more careful diagnoses.

After examining very carefully all the literature which I have been able to get bearing on this subject, I have come to the conviction that we have as yet no trustworthy statistics on which the most painstaking investigator could base a just opinion as to the relative prevalence of cancer in the last three decades. The same criticism may be made about other very positively expressed opinions. Statements have been made as to the relative prevalence of cancer in hot and cold countries, among savage and civilized peoples, among meat-eaters and vegetarians, and on certain geological formations, which statements cannot be supported by anything like scientific testimony. The inherent difficulties in the way of getting correct data on a large scale in regard to tumors are so great that we cannot accept without scrutiny any statements which are based on doubtful statistics, or on the impressions of travellers, or even of surgeons in large practice. These difficulties may never be altogether overcome, but it may be possible, by getting the profession sufficiently interested, so to lessen the causes of failure as to get approximately correct results. Until that is accomplished we must be content to suspend our judgment on many questions. What we urgently need at the present time is, not hasty generalizations from limited experiences, but correct information, carefully and systematically acquired, as to the conditions which influence tumor growth.

A review of the numerous hypotheses and theories which have been advanced to account for the existence of tumors does not inspire the student with the feeling that the problem is near solution. Those which seem most rational, like Cohnheim's, and Ribbert's modification of Cohnheim's, impress one rather as exaggerated statements of certain facts than as serious attempts to explain them.

There can be little doubt that there are certain defects in development which are characterized by the formation of superfluous cells; that in some cases these cells suffer displacement; and that in some they persist and develop abnormally in intra-uterine or post-uterine life. The riddle is not solved by these statements, but is differently presented. We have still to learn the nature of that physiological bond which makes the existence of complex organisms possible—the bond which is broken whenever and wherever a tumor exists.

PARASITICAL RELATIONS OF CANCER.

By HARVEY R. GAYLORD, M.D., Buffalo, N. Y.

The belief that the cancerous process is due to some parasite has come down to us with our earliest knowledge of this affection. In the minds of the earlier observers this was due to the frequent confusion of cancer and certain of the infectious granulomata, especially tuberculosis. The clinical course of many of the sarcomata and the difficulty frequently met with in distinguishing sarcoma in its clinical aspect from such processes as Hodgkin's disease, which is undoubtedly infectious, have sufficed to keep alive, in the minds of many clinicians, the belief in the infectious nature of the malignant processes. It is obvious that a purely clinical point of view may be one-sided, but there is little doubt that many of the theories which have been advanced by pathology have not sufficiently considered the clinical aspects of the disease or else have ignored them entirely. The majority of pathologists are at present distinctly opposed to the belief that any parasite exists which could fulfil the rôle of a parasite for cancer. It is obvious that no ordinary parasite could fulfil this rôle. Therefore, when, in 1886, Scheuerlin, and later Schill, detected bacteria in cancer, it was not long before these organisms were found to be simply harmless saprophytes. This also may be stated to have been the case with the yeast organisms or blastomycetes, which have been more recently described by San Felice and others as occurring in carcinomata.

INCLUSIONS IN CANCER.

Since the earliest histological investigations of cancer, there have been observed in the cells certain objects, as to the significance of which much discussion has taken place. It is not profitable to consider here the question whether or not these bodies are parasites. It is interesting to note, however, that as early as 1847 Virchow described these objects, believing them to be metamorphosed nuclei or degenerative changes, of a fatty character, in the protoplasm of the cancer cells. They were again described in 1889 by Thoma, who believed that they were protozoa; in 1890 by Sjöbring and Siegenbeck van Heukelom; in 1891 by Steinhaus; in 1892 by Soudakewitsch, Borrel, Foa, Kursteiner, Podwyssozki, and Sawtschenko;

in 1893 by Ruffer and Walker and Ruffer and Plimmer; in 1894 by J. Jackson Clarke and Cattle; in 1896 by Pianese; in 1898 by Bosc; in 1901 by E. van Leyden and Gaylord; in 1902 by Feinberg, Greenough, Nösske, and Posner; in 1903 by Apolant and Embden; and in 1904 by G. N. Calkins. Of these observers, Pianese, Greenough, Nösske, and Apolant and Embden believed that the bodies in question are not parasites. The others held them to be protozoa or allied organisms. Calkins holds that, although they have not been proven to be so, they may nevertheless be parasites. The forms in question have come to be known as "Plimmer's bodies," or Van Leyden's "bird's-eye inclusions," or the "*x*-bodies" of Behla. They are spherical structures, which vary in size from four to forty microns. They have a delicate limiting membrane and a central, highly refractive body. The space between the central body and the margin sometimes contains a fine protoplasmic structure, while at other times granules are regularly distributed between the periphery and the central body. They have been observed in the nucleus and in the protoplasm, and in the intranuclear forms they present an appearance not unlike the similar inclusions which have been observed in smallpox and in vaccinia. These bodies have been seen in the fresh state, but they are best demonstrated by complicated hardening and staining methods.

There is no direct proof that these bodies are parasites, although many observers have maintained the belief that they are such. On the other hand, those who have attempted to show that they are not parasites have been forced to employ the same methods of reasoning, and it can be fairly stated that to-day neither those who hold that they are of a parasitic nature nor those who hold that they are not, are in a position to prove their contention. The preponderance of opinion is opposed to the view that these bodies are of a parasitic nature, but this is, to no inconsiderable extent, due to the fact that the majority of pathologists hold, on *a priori* grounds, that cancer is under no circumstances an infectious process. There are some observers, however—notably Borrel—who hold that cancer is an infectious process, that these inclusions are not parasites, but that there is an infective agent in cancer which is either undemonstrable or ultra-microscopic. Perhaps the best arguments in favor of the inclusions being parasites are these: Their similarity in appearance to a known organism—Plasmodiophora brassicæ—and the fact that in certain respects they resemble certain forms of the smallpox organism.

CANCER AND THE ACUTE EXANTHEMATA.

Although at first thought there would scarcely appear to be any relation between the cancerous process and the acute exanthemata, yet this analogy between the two groups of diseases has been strongly advocated, principally by Bosc, Gaylord, Borrel, and von Wasielewski; the first two observers basing their

advocacy on the ground of the similarity of some of the inclusions in the two processes, and Borrel and von Wasielewski on more general grounds.

It will perhaps be of interest to follow more closely the relation which exists between the two processes. Those who discovered a resemblance between the inclusions found in cancer and those observed in smallpox and vaccinia were the first to call attention to the analogy between the two processes. It was Gorini, namely, who first detected points of similarity between certain larger forms of the vaccine body as they appeared in the inoculated corneas of rabbits and the cell inclusions of cancer. This similarity applies only to certain larger forms of the vaccine body which had been previously described by L. Pfeiffer, Guarnieri, and Clarke; but Gorini was able to trace a gradual transition between the larger typical vaccine bodies and these larger inclusions, which resemble the inclusions in cancer. In 1900 the writer observed a similarity between certain of the cancer inclusions and certain forms of the vaccine organism, and from this observation it was inferred by him that if the inclusions in vaccine were parasites, then in all probability the inclusions in cancer were of the same nature. On the same day of the same year Bosc published an article in which he advanced exactly the same idea. At the same time he called attention to the fact that in the lesions of sheep-pox also there were bodies which bore a close resemblance to some of the cancer inclusions. Sheep-pox is characterized by the development of both epithelial and connective-tissue nodules in the subcutaneous tissue. Bosc found, in the exudate from fresh pustules, characteristic epithelial cells containing highly refractive bodies surrounded by a clear zone of protoplasm; in other words, inclusions closely resembling those described in the epithelial cells of cancer. Similar inclusions were found in the cells forming the connective-tissue nodules. A sheep's cornea inoculated with the virus of sheep-pox presented lesions very similar to those resulting from the inoculations of the rabbit's cornea with vaccine, and Bosc believed that sheep-pox represented an infection lying midway between the malignant epithelial processes and the infectious exanthemata. It is unnecessary to state that the parasitic nature of these inclusions cannot be proved by histological methods alone; and the experiments thus far made with cancer have failed to bring any proof of its specific qualities. On the other hand, the work of Councilman and Calkins and of Bosc and Howard has again brought the significance of the vaccine and variola inclusions into the foreground, and it must be recognized that if these last inclusions—which are apparently incapable of cultivation and which are demonstrated by methods similar to those employed in the case of cancer inclusions, but which present more specific characteristics than do the latter—are ultimately shown to be parasites, then there is a prospect that future investigation may show that the inclusions found in cancer are also of the same nature.

Arguments in favor of the parasitic factor in cancer can, however, be adduced without the aid of these inclusions.

GENERAL ARGUMENTS IN FAVOR OF THE INFECTIOUS NATURE OF CANCER.

Transplantation Experiments.

Experimental methods in cancer research have opened a new era. This has been made possible by the discovery of the transplantability of tumors in animals of the same species, the first extensive demonstration of which we owe to Hanau, who succeeded in transplanting to the third generation a carcinoma of the rat. Before Hanau, however, as early as 1875, Nowinsky succeeded in transplanting a medullary carcinoma taken from the nose of a dog, successfully in two out of forty-two inoculated dogs. Wehr in 1883 succeeded in transplanting a medullary carcinoma from the vaginal mucosa of a bitch into a number of dogs. Most of these tumors retrograded, but in one animal the tumors grew to considerable size and produced metastases in the adjacent lymph nodes. Following Hanau, Morau in France, Leo Loeb in America, Jensen in Copenhagen, Borrel in Paris, Ehrlich in Frankfurt, Bashford in London, and the New York State Cancer Laboratory in Buffalo have all experimented with the transplantation of primary tumors—mostly in mice, Loeb's first observations being on a sarcoma of the rat. The extent to which this work is now being carried on can be appreciated when it is stated that one tumor alone, that of Jensen, is now being worked upon in at least seven laboratories, and that this tumor has been transplanted to somewhere near the eightieth generation.

The attention which has been attracted to the occurrence of primary tumors in mice has led to the discovery of a large number. Thus Ehrlich has succeeded in collecting tumors in 154 white and 10 gray mice; Bashford collected 9; Loeb has recently detected a spontaneous tumor in a mouse; and the New York State Laboratory is in possession of 8 primary tumors. Borrel has secured in Paris 30 examples of spontaneous tumors in mice, and Haaland speaks of 62 cases known to the authorities of the Pasteur Institute. The latter authority calls attention to the fact that the 62 spontaneous tumors observed in Paris were all in elderly females, and that all of the tumors were adenocarcinomata, involving the abdominal aspect, the axillæ, the groins, or the neighborhood of the anus or the vulva of these mice. They were all derived from the breast. Ehrlich likewise calls attention to the fact that, of 164 spontaneous tumors observed in his laboratory, all occurred in aged females and were all positively derived from the mamma. Eight out of nine of Bashford's mice were elderly females, and the tumors were likewise all derived from the breast; in the one exceptional case—that of a male—the tumor was situated near the root of the tail and presented the same characteristics as the other tumors. Of the eight tumors observed in Buffalo, seven were in females, the sex of the eighth having been unfortunately overlooked. They all presented characteristics similar to those observed in the recognized adenocarcinomata derived from the breast in the mouse.

The fact that all of these tumors were derived from the breast, and the further fact that the mouse appears to be much more frequently affected by carcinoma than are other small animals, can only be explained, as Ehrlich has pointed out, by the facts that all of these mice were obtained from dealers who were engaged in raising white mice for the market and that all the females are employed for breeding purposes. The fact that almost all of the tumors have appeared in elderly females certainly points to the probability that the tremendous demands made upon the mammary tissue of these animals explain the almost exclusive appearance of this form of the tumor. In connection with these facts, the observation of Borrel that healthy mice, when kept for a sufficient period of time in the same cage with infected mice, may develop spontaneous tumors, is of the greatest importance. It has likewise been observed that wherever one spontaneous tumor developed in any particular locality where the mice are being bred, either simultaneously or later, mice with similar tumors have been found.

Communicability of Cancers in Mice.

The most striking example of the endemic occurrence of cancer is described by Borrel, who, in the course of two years, observed in one breeding place twenty cases of carcinoma of the breast. All of these mice had, at one time or another, been in the same cage. He observed further, in a second case, in the course of one year, five or six cancer mice, all of which developed in one cage. A similar endemic occurrence of cancer in the rat was observed by Hanau, who first successfully transplanted cancer in this animal. He observed in the course of six years three cases of squamous epithelioma of the vulva. There were in all about one hundred rats, all the offspring of two pairs. Perhaps the most striking evidence of cage infection is found in an observation recently made in the State Cancer Laboratory, combined with a previous observation made by Leo Loeb. Loeb states that in January, 1900, there developed in a group of cages containing rats in the Chicago Polyclinic Laboratory a spontaneous sarcoma of the thyroid. In November of 1901 a second case of sarcoma of the thyroid developed in the same group of cages, and in the autumn of 1903 a third case. The rats had been moved about from cage to cage and were all the offspring of a certain limited number of rats. The tumors presented identical histological characteristics. The first and second rat tumors were used for transplantation, in both cases successfully. On transplantation the tumor presented the characteristics of spindle-celled sarcoma, which in many animals produces characteristic regional and organal metastases. Sections of this tumor have been repeatedly shown at scientific meetings, and there is absolutely no question as to its being a genuine spindle-celled sarcoma. The spring and summer of 1902 were spent by Dr. Loeb at the State Cancer Laboratory in Buffalo. He was provided, for the accommodation of his animals, with two large and a number of small cages. He brought with him a number of rats which had been inoculated from his second sarcoma of the

thyroid obtained in Chicago. During the period of his stay in Buffalo ne carried out a number of successful transplantations. On leaving the laboratory in September he took with him a number of rats with tumors, but these became infected, and later the tumor was so infected as to be no longer transplantable. After Dr. Loeb's departure all rats were removed from the laboratory. The smaller cages were sterilized in the hot-air sterilizer, but the two larger cages which he had employed, being too large for such sterilization, were simply cleaned and put away. For a period of several months after Dr. Loeb's departure there were no rats of any kind in the laboratory. In the summer of 1903 some rats were purchased in Buffalo for other purposes than tumor transplantation, and a number of them were placed in the two large cages mentioned. In the spring of 1904 there was found in one of these cages a rat with a tumor the size of a horse-chestnut in the subcutaneous tissue of the right abdominal aspect. This tumor was removed by operation, and proved to be a fibro-sarcoma. It was transplanted to other rats, but without success. The occurrence of the development of this sarcoma in the rat was noted and the cage was marked. There were then introduced into the cage a number of adult rats, but, owing to an epidemic of itch among them, it was found necessary to remove the cages containing them to the basement to prevent the possible spread of this infection to the hundreds of mice which occupied the regular animal space in the laboratory. During the summer of 1905 there were found in this cage two adult rats, both males, one with a large fibro-sarcoma in the right abdominal aspect and the other with a large sarcoma of the thyroid. The latter rat died early in October. Sections of the tumor showed it to be identical in appearance with the three primary sarcomas of the thyroid described by Loeb, which developed in the cages in the Chicago Polyclinic Laboratory. In the middle of October an operation was performed upon the other rat. Sections showed that the tumor was a fibro-sarcoma of identical appearance with the one which had appeared in the cage a year before. A number of rats had died during the course of the summer with tuberculosis, so that at the time of the development of the tumors there were but four adult rats in the cage, the two with the tumors and two without. No other tumors have developed in rats in any of the other cages in the laboratory, although the small cages employed by Dr. Loeb and subsequently sterilized have now had rats in them for a period of two years. Aside from the three cases of primary sarcoma of the thyroid developed in Chicago and described by Dr. Loeb, during the period of three years since his departure from the laboratory with his inoculated rats no other author has described sarcoma of the thyroid in the rat, and none has been known to develop in any of the establishments in which these animals are bred. The demand for animals with tumors has become so great that all breeders of white mice and white rats are now on the lookout for tumors, so that the possibility of their having been overlooked is reasonably remote.

Haaland calls attention to a case in which a woman in Paris purchased two white mice for breeding purposes. In the course of two years she sold about two hundred young offspring, reserving the mature mice for breeding purposes. Among these she observed twenty spontaneous tumors. The last three of these mice, with the cage in which they had developed their tumors, were brought to the Pasteur Institute. The mice were removed from the cage and were placed in a new cage, and into the cage in which they had developed their tumors were placed new mice. None of the mice which had previously been in the cage in which the tumor developed, or the new mice which had been placed in it, developed tumors under subsequent observation. The three mice, however, which had already developed sporadic tumors were placed in a new cage, and in this cage were placed with them a number of mice derived from healthy stock, their ancestors so far as known never having had sporadic tumors. Of the healthy mice thus placed in contact with the mice already infected, four developed spontaneous tumors. From this it would appear that to a certain extent these mouse tumors are contagious. If this be admitted, what is the significance of the almost exclusive development of primary carcinoma of the mamma in elderly females among these mice? Ehrlich points out that two explanations are possible. First of all, by reason of the great fertility of the animals the older females are almost constantly carrying and nursing young. It is therefore reasonable to assume that the tremendous demands made upon the breast predispose to an unlimited proliferation of the epithelium of that gland. On the other hand, it is probable that, through the indiscriminate nursing of the young, first by one mother and then by another, an infection of the breast in one mouse might easily be transferred to that of another mouse. It has been shown that in the early stages of carcinoma the breast still possesses the power of lactation, and it is therefore perfectly possible that, through eversion of the nipple, the virus may be transferred from that structure in one animal to the corresponding structure of another. Both in Paris and in Ehrlich's laboratory careful experimentation is being carried on for the purpose of ascertaining whether or not this occurrence can be experimentally proven. In the light of Borrel's observation—viz., that healthy mice which are brought in contact with infected mice can acquire these tumors—it would seem that the exclusive appearance of tumors of the breast among animals used solely for breeding purposes presents very suggestive evidence in favor of an infectious factor.

Transference of the Infectious Factor in Cancer Cells to Normal Epithelium.

The evidence thus far adduced applies only to primary tumors. If there is a contagious factor which can be transferred from one animal to another, bringing about the transformation of normal epithelial cells into cancer cells, then it is not improbable that, in the very beginning of cancer, this contagious factor may be

transferred for a limited period from one cell to the next. In fact, pathologists generally recognize that, in small, beginning carcinomata, such a transformation can be observed. We have from Orth, in his most recent utterance on this subject, the following: "I am, I confess, of the opinion that there are cancers in which the transformation of preformed epithelial cells into cancer cells takes place continuously in the tissue bordering upon the margin of primary tumors; also that there are multicentric cancers, not only in the sense that the cancer change takes place at the same time in different neighboring spots, but also in such a manner that one spot becomes cancerous later than another." If a primary cancer starts from a given centre and the cancerous transformation spreads from cell to cell, it must be that that force or factor which endows normal epithelium with the power of limitless proliferation is transferred, at least for a certain period of time, from the involved cells to the adjoining normal ones. Although this appears to be the case in the period of the inception of a cancer, experimentation has shown that the cancer cell, once endowed with this power of proliferation, retains it most persistently, and a transference of this power to other cells never occurs, unless one or two suggestive observations, which will be referred to later, are evidences of such transference.

Success in transplanting these sporadic tumors in the mice has been variable, but the general experience tends toward more successes as the work progresses. In six of the mice in which cancer was transplanted by Bashford, there were only two in which the disease persisted beyond the second generation. Borrel is in possession of an epithelioma and an adenocarcinoma which are transplantable, and Ehrlich has at present ten different sporadic tumors in process of transplantation, some as advanced as the sixtieth generation. Bashford has had over three thousand transplanted tumors under observation. Ehrlich's, Borrel's, and Jensen's observations must likewise run into high figures, and the New York State Laboratory has already had about six hundred. It will thus be seen that the last two years have been very fruitful in experience in the investigation of cancer, and it may be said that, although the work has just begun, many ideas which we have held regarding this process have been shown to be erroneous, and many characteristics have developed which were entirely unexpected.

Characteristics of Transplantable Mouse Tumors.

It must be pointed out that the mere transplantability of cancer throws no light upon the mechanism by which spontaneous tumors develop. These transplantations are modifications of the process of metastasis. The success with which they have been accompanied has shown great variability, but on the whole the experiences of all laboratories have been that tumors which have been often transplanted acquire an increased virulence, so that, although the success attending the first attempts at transplantation has in many cases been as low as

one or two per cent, in later cases the virulence has risen to such an extent that the average has been as high as from eighty to one hundred per cent. A most interesting example of the tremendous virulence of these transplanted tumors is found in one which is under observation by Ehrlich. This, known as No. 7 in his series, presents a virulence which is most astonishing. The transplantations with this tumor material have for a considerable period of time given from eighty to one hundred per cent of successes. The transplanted tumor grows with such rapidity that in eight days after inoculation it has been found to weigh 2 gm.; at the end of two weeks, over 3 gm.; and at the end of three weeks, usually 5 gm. Tumors as large as the mouse itself not infrequently develop within two months from the time of inoculation. All laboratories which have been working on transplantation have had similar experiences. Some tumors are found to grow very slowly, as did that of Morau, which required months for its full development, whereas others present the characteristics of the tumor described above. In all tumors, however, repeated transplantation, instead of weakening the energy of the tumor, seems to increase its virulence, and it is now recognized that the most distinguishing feature of cancer is the unlimited power of proliferation which the cancer cells possess, this power having already carried some tumors beyond the sixtieth generation of transplantation through healthy mice.

All this experimentation has failed to show us how the cancer cells acquire this phenomenal power of proliferation. That the characteristic factor of cancer is found only in the epithelium is shown by the fact that the stroma in the transplanted tumors is furnished by the host. That this factor, in the course of transplantation of mouse tumors, is occasionally transferred to the connective-tissue elements of the stroma, endowing them with sarcomatous characteristics by which the tumor is transformed into a mixed tumor, is shown by the fascinating publications of Ehrlich and Apolant (*Berl. klin. Wochenschr.*, 1905, No. 28, and 1906, No. 2). These observers have now encountered this phenomenon in three tumors. In the first case observed, the tumor presented the usual characteristics of the adeno-carcinoma of the mouse and had been transplanted without any change to the sixth generation. The tumor consisted of nests of varying sizes of alveolar arrangement, with a not very well developed connective-tissue stroma. Between the sixth and ninth generations the tumor underwent a change in which the carcinoma suddenly presented the characteristics of a mixed tumor, the thin connective-tissue stroma presenting every evidence of active proliferation; wide avenues of closely packed, deeply staining spindle cells, with abundant karyokinetic figures, appearing between the nests of epithelium. These characteristics persisted from the ninth to the thirteenth generation, the epithelial characteristics gradually diminishing and the nests becoming smaller and more widely separated; and in the fourteenth generation the epithelium had entirely disappeared from the tumor, leaving a spindle-celled sarcoma, which is still being transplanted and has reached the fortieth genera-

tion. The accompanying table from Ehrlich will serve more graphically to emphasize this remarkable observation:

Family 16 Carcinoma
1 Generation Carcinoma
2 Generation Carcinoma
6 Carcinoma
9
Mixed tumor 10b — 10a Mixed tumor
11
12a — 12b — 12c mixed
13a Sarcoma — 13b Mixed — 13c Mixed
14 Sarcoma
15 Sarcoma

The generations marked a, b, c descended from various mice of the preceding series.

Recently Apolant and Ehrlich have reported two further similar observations. In one of these the sarcomatous transformation developed in an adeno-carcinoma which was produced by mixing together various adeno-carcinomata which were respectively in the twenty-first, the thirty-third, the twenty-third, and the nineteenth generations of transplantations. One of the strains derived by this mixture, between the twelfth and fourteenth generations, showed a marked increase in the proliferation of the connective-tissue stroma, which awakened at once the suspicion that the development of sarcoma was taking place. In the sixteenth generation this was so far developed that the tumor presented the characteristics of a mixed tumor. In contrast to the first case reported, the differentiation between the nests of epithelial cells and the proliferating stroma was not nearly so marked as in the preceding case. The sarcoma cells likewise were more polymorphous in appearance, typical spindle cells forming only a part of the tumor. They lay in irregular masses that filled the spaces not occupied by the net-like structure of the epithelial portion of the tumor. The proliferative characteristics of the sarcomatous portion of the tumor were not nearly so marked as in the first case. The tumor at present is in its tenth generation of transplantation, and the proliferation of the connective-tissue and epithelial elements appears to be about on the same footing as it was before, the tumor having during the last six months shown but

slight changes in the relative proportion of epithelium and connective tissue. The rapidity of growth of this tumor shows no diminution, the authors having observed tumors of enormous size, in many cases equal to that of the mouse itself.

The third observation is the most striking of all. It occurred in the course of transplantation of Ehrlich's tumor No. 7, which is the most virulent of all mouse tumors yet under observation. This tumor had shown, from the fortieth to the sixty-eighth generation, a marked increase in the connective tissue without the stroma presenting the characteristics of a sarcoma, when suddenly in the sixty-eighth generation it took on a marked sarcomatous appearance, associated with such colossal proliferation that in the next generation many of the tumors were sarcomas without any evidence of epithelioma. Here and there some of the transplanted tumors contained a few nests of epithelium. These remnants of carcinomatous epithelium have been detected as late as the seventy-first generation. The sarcoma cells in this tumor were likewise more polymorphous in character, those of a spindle shape being in a minority. This tumor has now been transplanted three generations further without any loss of the colossal proliferative qualities with which it has been endowed from the first. The explanation of this phenomenon given by Ehrlich is that some form of stimulus present in the carcinoma cells is in certain phases of its development transferred from the epithelium to the connective-tissue stroma of the tumor and transforms the connective-tissue cells of this structure into typical sarcoma cells capable of probably indefinite transplantation.

It is impossible to draw conclusions from a single observation, but the phenomenon described above may possibly be explained by the assumption that the chief characteristic of a cancer—viz., its power to proliferate to an unlimited extent—has, in this particular instance, been transferred from the epithelium to the connective tissue. To assume, on the other hand, a transformation of epithelial cells into connective-tissue cells, would be contrary to all our histological knowledge. It can, of course, be said that the phenomenon under consideration represents merely the sporadic development of a sarcoma on the basis of a carcinoma. That the *x*-factor in cancer may possibly be transferred to other cells is shown by the frequent observations referred to by Haaland, and observed in Buffalo, of the development of primary adenomata in the lungs of mice which have been the subject of transplantation of these mammary tumors. Haaland refers to the fact that this primary development of adenomata in the lungs of mice is a not uncommon occurrence, and our own observations corroborate this statement.

Natural Immunity to Implantation in Mice.

It has been found in all laboratories that a certain proportion of mice cannot be inoculated with the tumor. Thus far, a natural immunity against these inoculation experiments appears to bear no definite relation to heredity, but in all

laboratories mice have been found which appear to be permanently immune, and these mice have frequently been the offspring of parents both of which were afterward successfully inoculated and died of the tumors.

Spontaneous Retrogression in Cancer of the Mouse.

Although the disease, once established by implantation, is in a very large per cent of the cases fatal, in all laboratories occasionally spontaneous cures have occurred. These have been observed in Ehrlich's laboratory and also by Bashford; and apparently, up to the present time, the greatest number have occurred in the Jensen mice under observation in the State Laboratory in Buffalo. Immediately following the transplantation of these tumors—which is done by taking uncontaminated tumor, mixing it in a mortar with three or four parts of normal salt solution, and injecting it beneath the skin of the back through a coarse needle with a syringe or introducing particles through a small trocar—there is frequently a slight reaction, which subsides on the second or third day. It is obvious that in many of these experiments transient infection occurs, as shown by the formation of an abscess. This usually interrupts the experiments but occasionally the swelling subsides and ultimately a tumor develops.

In the period from February to June, 1905, not less than twenty per cent of the tumors resulting from successful inoculation underwent spontaneous retrogression. This is a higher percentage of spontaneous recoveries than has yet been reported from any other laboratory. The distribution of the period in which the processes of retrogression were apparent shows that more spontaneous retrogressions occurred early in the process than late. There are, however, a certain number of retrogressions which occurred in what would normally be the last stages of the disease. One of the most striking examples occurred in a rapidly growing tumor, which reached a weight of over 3 gm. in forty-three days after the inoculation, then began to retrograde and ultimately disappeared.

That a spontaneous cure of a genuine carcinoma in the mouse should occur and should be well authenticated would at first seem surprising, but a careful review of the literature has shown that undoubted cases of spontaneous cure have also been observed in human beings. It is natural that a greater percentage of these cures should occur under experimental conditions than under the conditions in which we encounter cancer at the bedside. Mice used for experimentation are taken at random, and it is obvious that some of them have a greater resisting power than others, as shown by the fact that a certain percentage of them possess a natural immunity which protects them from being successfully inoculated. The cases which we meet clinically are those of individuals who apparently have no sufficient immunity, and we see therefore only the unfavorable cases. It is not improbable, however, that even in human beings patients become infected with cancer, but make early spontaneous recoveries, perhaps without attracting even their own attention.

Evidence of an Acquired Immunity against Cancer in Mice.

Researches in the State Laboratory as to the nature of the phenomena associated with spontaneous cure point very strongly toward the presence, in mice which have recovered spontaneously, of a form of acquired immunity. This is shown by the failure successfully to reinoculate any mouse which has spontaneously recovered. The immune factor is apparently present in the blood, and in some mice has been sufficiently active, when injected into other mice with growing tumors, to influence the growth of the tumor. In this way small tumors have been made to retrograde and large tumors have been inhibited in their growth. Further proof of the presence of an immune factor in the blood of mice is found in the recent observations of Clowes, which show that when cancer material is treated with a sufficient proportion of the blood of spontaneously recovered mice the number of successful inoculations is markedly reduced.

Histological Characteristics of Retrograding Mouse Tumors.

Examinations of the histological appearance of tumors undergoing spontaneous retrogression, and of those retrograding under the influence of injections with immune serum, show identically the same picture. If the action of this

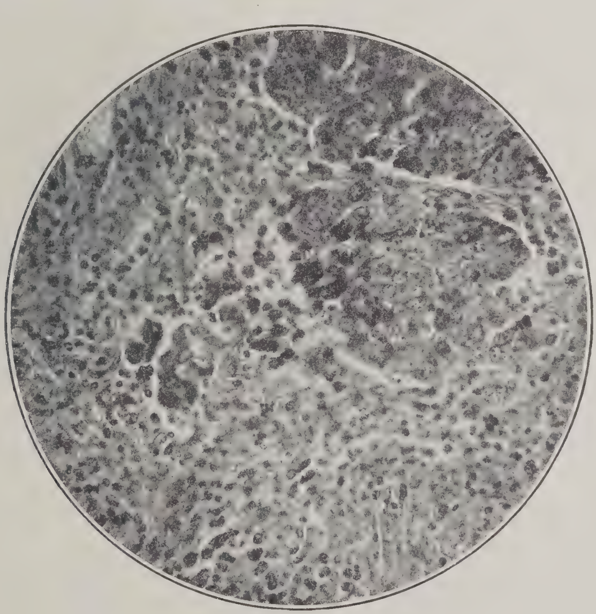

Fig. 119.—Microphotograph. × 260. Epithelium at Margin of Tumor Undergoing Retrogression from *x*-ray Treatment.

serum were cytolytic in its nature, we should expect to find evidences of destruction or direct injury to the cells, but this is not the case. About the margins of retrograding tumors one finds that the cells have undergone simple atrophy, and that where groups of cells remain they frequently coalesce into pseudo-giant

cells. These are surrounded by connective tissue, and ultimately, through the process of atrophy, disappear. In tumors undergoing retrogression hemorrhage is a frequent occurrence. An examination of the cancer cells immediately adjacent to the hemorrhages in the tumor shows that this process of simple atrophy is most marked where the cells have come in contact with the extravasated blood. Practically, one can see here the direct action of the blood upon the cells. There is no necrosis of the protoplasm, and karyokinetic figures can be found in the epithelial cells until the very last. The picture presented shows that the epithelial cells are subjected to a process which is identical with that which overtakes transplanted or misplaced normal epithelium. Leo Loeb and others have

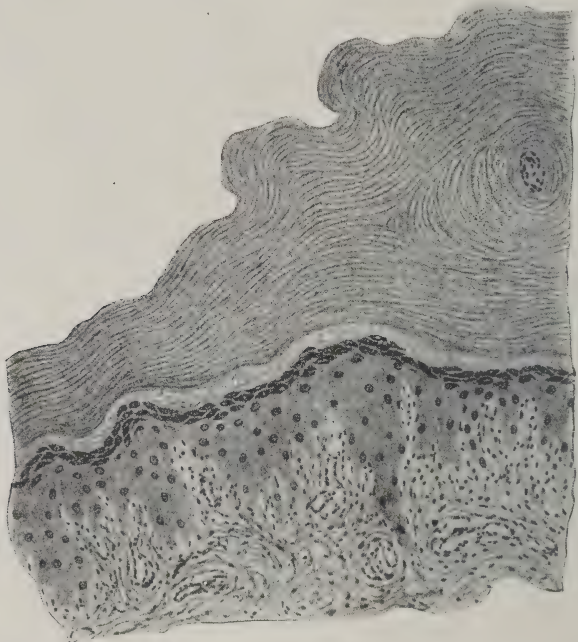

Fig. 120.—Section of Wart Thirteen Days after First Treatment, Nine Days After Last Treatment with x-ray, Showing Complete Hornification of Epithelium of Wart and New Skin Formed from Deeper Layers. (*Perthes.*)

shown that if fœtal epithelium is aseptically transplanted into the subcutaneous tissues in adult animals, it is able to maintain itself for a period of time during which its dynamic force suffices for proliferation to the sixth or seventh generation, after which the force is expended, the cells undergoing atrophy and becoming surrounded by connective tissue, which grows between them. The picture presented here is exactly like that found in these retrograding tumors.

From this observation it must seem obvious that in spontaneously retrograding tumors the immune factor, instead of working directly upon the cells, reduces them to the status of normal epithelium, and they are then removed by a process of atrophy and repair which is identical with that which overtakes misplaced normal epithelial elements. Becher, Petersen, and Schwartz have shown that

similar reparative processes are frequently at work in many human carcinomata. That the connective-tissue activity is secondary is shown by interference with the immune mechanism, which interference can be brought about by bleeding. In the New York State Laboratory it has been found that in the case of tumors which were undergoing retrogression as the result of injections of immune sera, severely bleeding the mouse would interrupt the process and the tumor would thereupon begin to grow as rapidly as ever. This observation, in connection with the facts which tend to show that the immune factor is in the blood, strongly

Fig. 121.—Section of Untreated Wart for Comparison. (*Perthes.*)

indicates that the proliferation of the connective tissue is but a secondary process, which only becomes active when the cancer cells are reduced to the status of normal epithelium.

Identity of Histological Characteristics of Spontaneously Retrograding Tumors and Tumors Retrograding through Treatment with Immune Sera, the x-Ray, or Radium.

The changes which are brought about in carcinoma, either in man or in animals, by exposing them to the activities of the x-ray or of radium, have been shown by Exner, Perthes, and others in man, and by Apolant and Embden and Bashford in mice, to present exactly the same histological picture as that which

is presented by tumors undergoing spontaneous retrogression. This fact has been under observation for over a year in Buffalo. A section of a tumor retrograding under the activity of the x-ray or of radium is in no way distinguishable from one taken from a tumor undergoing spontaneous retrogression or retrogression induced by serum treatment. Examination of the blood of mice which have recovered from tumors through the activity of the x-ray shows that this fluid does not contain any acquired immune factor. If, however, in the course of treatment a mouse is heavily bled, the tumor will frequently begin to grow—a phenomenon which leads to the conclusion that the x-ray does not act directly upon the tumor, but through such immune factors as the mouse still possesses.

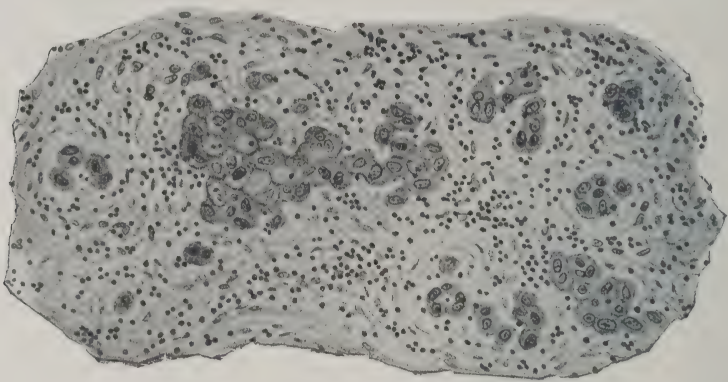

Fig. 122.—Human Tumor Undergoing Retrogression from Treatment with x-ray. (*After Perthes.*)

For this reason it would appear that the x-ray and radium reduce the virulence of the tumor or so injure the x-factor that the natural immunity brings about the retrogression of the tumor. In this way it is possible to explain those tumors which are not affected by the x-ray, and also the fact—which has been frequently observed—that tumors which are being favorably affected suddenly begin to grow in spite of continued treatment.

Significance of Perthes' Experiments with the x-Ray on Warts.

That the x-ray, either directly or, as would appear, indirectly, robs the epithelial cell of the factor which causes its unlimited proliferation, and leaves the normal epithelial cells unaffected, is shown by the interesting experiments on warts by Perthes. Perthes has clearly demonstrated that the dose of x-ray required in the treatment of cancer, or for the removal of warts, does not injure directly either the normal epithelium or the epithelial cells of the tumor. If the tissues surrounding the tumor are overdosed a so-called x-ray burn may be induced, but this is an injury entirely independent of the ideal therapeutic activity of the agent. Perthes has shown by sections that a wart which has been properly dosed, frequently with but one treatment, will thereupon undergo a process of retrogression, in which all of the cells forming the wart become horni-

fied, *with the exception of those of the deeper or germinal layer; and these promptly proliferate and produce, not a new wart as before, but normal new skin to repair the defect.* If the dose is not sufficient the superficial cells will undergo hornification, the wart will be reduced in size, but the cells of the deeper layer will again proliferate and produce a new wart. *This proves conclusively that the x-ray does not act through any form of injury to the cells themselves. It removes from them the tendency to proliferation which produces the wart, and leaves behind, in the necessary cells of the germinal layer, normal uninjured cells which are capable of producing*

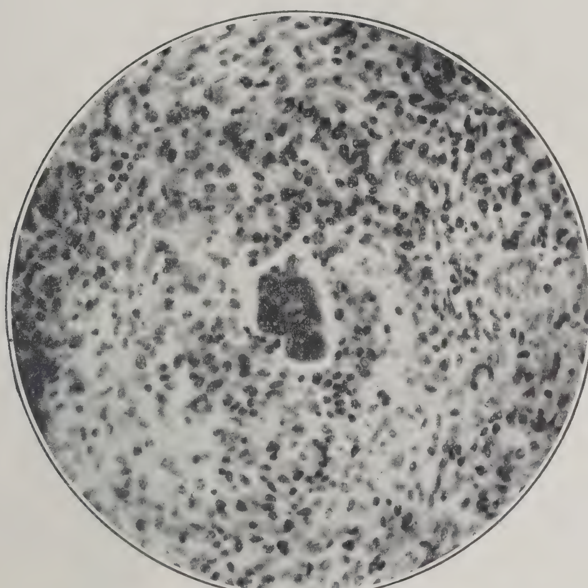

FIG. 123.—Microphotograph. × 260. Last Remnant of Epithelium from a Tumor Undergoing Spontaneous Retrogression.

new and normal skin. As it is the fate of superficial epithelial cells of the skin, when their period of utility is passed, to undergo hornification, the process of hornification in the cells of the wart is probably secondary. Once their power of abnormal proliferation has been removed, they succumb to that fate for which they were normally intended, which is hornification; and this involves all of the cells of the wart except those which are destined to resume their normal functions.

Characteristics of the Unknown Stimulus in Cancer.

The observations thus far accumulated on the spontaneous retrogression of tumors, the retrogression of tumors through an immune agent, and the direct or indirect activity of the x-ray and radium, tend to show that in these agents we have a means of removing from the cancer cell the x-factor. If, as conceded by Orth, there takes place at the margin of tumors a gradual transformation of normal epithelial cells into cancer cells, and if, by the action of immune sera and the x-ray, we can again reduce these cancer cells to the status of normal cells, it seems

almost conclusively shown that there can be added to a normal epithelial cell a factor which is capable of endowing it with the power of continuous proliferation, and which can again be removed from it, leaving a normal epithelial cell. This normal epithelial cell, it is true, may be superfluous, in which case it will undergo processes of atrophy and removal the same as may take place in any other misplaced normal epithelial cell. But if, as in the case of warts, the cell still has a function to perform, it can resume its natural proliferative activity—an activity which does not overstep the bounds set by the physiological laws of normal life.

It has been suggested that the unknown factor in cancer is of a chemical nature. No less an authority than Marchand has suggested that it might be some toxin. If the facts in the case are considered, it is obvious, as Clowes has

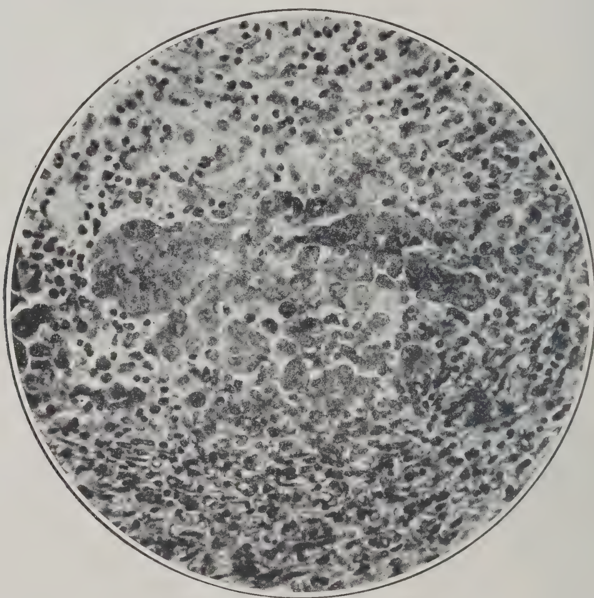

Fig. 124.—Microphotograph. × 260. Alveolus at Margin of Tumor Undergoing Retrogression from Serum Treatment.

shown, that this is impossible. An agent which is capable of keeping up continuous proliferation in cancer cells—which, theoretically speaking, starts with one cell and passes into its offspring through thousands of generations, during which time the number of cancer cells increases indefinitely—must, quantitatively speaking, increase in bulk. That this must be so is evidenced by the fact that this factor can be removed and must be removed from each and every cancer cell before the cell undergoes retrogression. This is shown to be the case in experimental tumors which are undergoing retrogression after treatment by the x-ray or through the activity of sera. The changes brought about by either of these agents is found to be most marked at the periphery of the tumor, and it has been found that epithelial cells taken from the centres of tumors which are

retrograding at the margins can be transplanted and will produce tumors, whereas the cells at the margins present changes which show that this would be impossible. Therefore, the agent must be removed from each and every cancer cell; and as this agent, although present in the beginning in but one or two cells, later comes to occupy a bulk of cells which can scarcely be estimated, and as, furthermore, it must likewise have increased rather than have diminished in activity, it must certainly also have increased in amount. How enormous the proliferative powers of even a small mouse tumor may be is indicated by the astonishing figures which Ehrlich has published in connection with his rapidly growing tumor No. 7. He has estimated that this tumor is now growing at a rate which would permit of its being carried through sixty generations in one year. As the tumor is now giving nearly 100 per cent of increase, he estimates that if from 12 to 15 mice were used for each transplantation, within one week 10 tumors of the size of that used for transplantation would be produced. From each of these in eight days 10 more could be produced, so that in the third generation 1,000 tumors, in the fourth 10,000 tumors, and so on, would result. If this were carried to the sixtieth generation, it would represent 10^{60} c.cm. of tumor if each tumor weighed but 1 gm. In the course of one year this would lead to a bulk of tumor which is scarcely comprehensible. It represents, according to Ehrlich, a cube the edge of which would measure 1,000,000,000,000 kilometres—a distance which it would require light 105 years to traverse. In spherical form it would represent a mass with a diameter 890 times greater than that of the sun, and a volume exceeding that of the sun 7×10^{26}. If the agent which could keep pace with this tremendous increase in bulk were a toxin, it could only do so by reproducing itself; and the only possible mechanism by which this could be brought about would be by the agent acting upon the protoplasm of the new cell in such a manner as to cause it to produce its like. No chemical agent, however, with which we are acquainted, toxin or otherwise, and which is capable of bringing about a reaction in living protoplasm, causes this protoplasm to produce the same agent. On the contrary, the protoplasm produces, in all cases thus far known, an agent which is antagonistic to the first—in other words, some form of anti-body. For this reason it is impossible to conceive of any chemical agent endowed with the power to fulfil the conditions of the x-factor. We are therefore compelled to assume that the x-factor must be some agent which can reproduce itself, and thus far the only agents with which we are acquainted which can accomplish this are living agents. Hence the most rational explanation of the unknown factor in cancer is that it is some living agent. If we so wish we can speak of this agent as a virus, as does Borrel, inasmuch as we do not know its specific nature. Borrel believes that there is an infectious factor in cancer as yet undemonstrated, and that it is in all probability an invisible or ultra-microscopic organism. The same contention has been made in the case of syphilis, because the agent was unknown (unless the recent observations of

Schaudinn, and of many others confirming it, should ultimately show that Spirochæte pallida represents a phase of the organism of syphilis); and it is likewise held to be true in smallpox, in vaccinia, and in other diseases.

Significance of Filtration Experiments.

The belief that the contagious factor is invisible has usually been based upon filtration experiments. The virus of sheep-pox has been shown by Borrel to pass through the Berkefeld and the coarser grades of the Chamberland filter. This has likewise been shown to be the case with vaccine virus. The question arises as to whether filtration experiments are necessarily an evidence of an ultramicroscopic organism. Borrel furnishes light on this point. In his filtration experiments with sheep-pox he discovered that when he diluted the virus with tap water, after four days there developed in the filtered and otherwise sterile virus a small protozoön, to which he gave the name Micromonas Mesnili. The organism when in its largest form in the virus—it is of course impossible to affirm that the organism under other conditions does not possess a still larger phase—was three or four microns long and as many wide. That the organism in question had nothing to do with the virus was shown when distilled or sterilized water was used to dilute the virus. Borrel found that his organism followed the same law as the active principle of the virus; that is, it passed through the filters through which the virus passed, and was held back by filters which were proof against the passage of the virus. That the organism passed through in some practically invisible spore form, and then developed on the suitable medium of the virus, was shown by its appearance in virus only after four days and the impossibility of detecting it in filtered water in which the larger forms did not develop. Borrel was forced to conclude: "Le passage à travers un filtre n'implique pas forcément l'idée d'un microbe invisible."

It must be noted that Borrel's Micromonas shows as its largest form an organism considerably smaller than the larger inclusions of vaccine, variola, and cancer. However, to assume that because the spore of an organism is sufficiently small to pass through a certain filter, its largest form would be within a certain limit of size, is not justified by biological knowledge. Calkins has described a protozoön, Lymphosporidium truttæ, the spores of which have a diameter of one and one-half microns and divide into six sporozoites, each less than one-half a micron in diameter. Borrel likewise points out that there is an essential difference between the smaller forms of motile animate parasites and bacteria of relatively the same dimensions. The first are more plastic and accommodate themselves to the pores of the filter, passing through where bacteria are held back. In all probability the sporozoites of Lymphosporidium truttæ, less than one-half a micron in diameter, would pass through a bacteria-proof filter; and yet the largest form of this organism is a multinuclear amœba twenty-five microns in diameter.

It will be noted that Micromonas Mesnili is three to four microns long and as

many wide. The Spirochæte pallida in its smallest form is one-quarter of a micron in diameter and four to fourteen long. Becchi has shown that even large protozoan amœbæ, twenty-five microns in diameter, may pass readily through Berkefeld bougies, and for this reason it is desirable to eliminate filtration experiments in attempting to determine the relative size of organisms. There is a not remote similarity between the subcutaneous lesions of syphilis and some of the infectious granulomata and even sarcomata.

Infectious Venereal Granuloma of the Dog.

In this connection Bashford has recently described the histological characteristics of an inoculable venereal granuloma found in dogs, which possesses certain characteristics of a malignant tumor, and presents still others which leave no doubt that it is an infectious process, although the virus or organism is as yet undetermined. This tumor seems to be almost a connecting link between the infectious granulomata and malignant tumors. It is common in the dog, is transmitted by coitus, and develops in the subcutaneous tissue about the genitals. The tumor cells are polygonal, with scanty granular protoplasm and large, spherical nuclei. In the resting stage they possess one large nucleolus and a delicate chromatin reticulum. Mitotic division is common in the nuclei, and, although the type is commonly bipolar, *multipolar figures* are not unusual. The tumor is divided up into lobules by delicate connective-tissue septa containing fully developed capillaries. Hemorrhages are frequently found. The general appearance closely resembles that of a round-celled sarcoma with a parenchyma arranged in alveoli. In primary tumors (see Fig. 125) Bashford shows that a transformation of the connective-tissue cells into tumor cells can be demonstrated. This is only apparent where the rapid growth of the tumor has not resulted in pressure upon the surrounding structures. One striking feature of the tumor is that, although this transformation is going on at the periphery, the greater portion of the bulk of the tumor is brought about by proliferation of the tumor cells, in this way closely resembling the method of growth of a true malignant tumor. In the later stages the tumor grows almost entirely from its own resources. The primary lesion in this tumor is therefore in no way different from that of a primary sporadic carcinoma, in which there is likewise a transformation, at the margin, of normal cells into cancer cells. When, however, this tumor is transplanted, its true characteristics appear. According to Bashford, when the tumor cells are implanted in the subcutaneous tissues of a new host, all of the implanted cells, instead of continuing to proliferate, disintegrate, and a new tumor is formed by the action of the specific factor upon the connective-tissue cells of the host. Evidence of this process is found by Bashford at the margin of newly developing nodules after implantation. Inasmuch as Bashford believes that the implanted cells all disintegrate, it is obvious that the virus is the only factor which persists.

All attempts to determine the precise nature of this virus have thus far failed. Filtration experiments do not appear to have been carried out thus far. These tumors frequently grow to great size and sometimes undergo spontaneous retrogression. In transplantation experiments new tumors can be recognized in from eight to ten days, and subsequently they attain a diameter of several inches. Metastases may likewise develop in the mesentery after intrascrotal inoculation, and the lymph nodes adjacent to large growths are frequently enlarged. The disease cannot be transmitted to the cat, rabbit, guinea-pig, or mouse. Bashford concedes that, in its histological features, local mode of origin, partial growth from its own resources (in the later stages causing pressure on surrounding tissues and organs), and in the limitation of its transmissibility to one species,

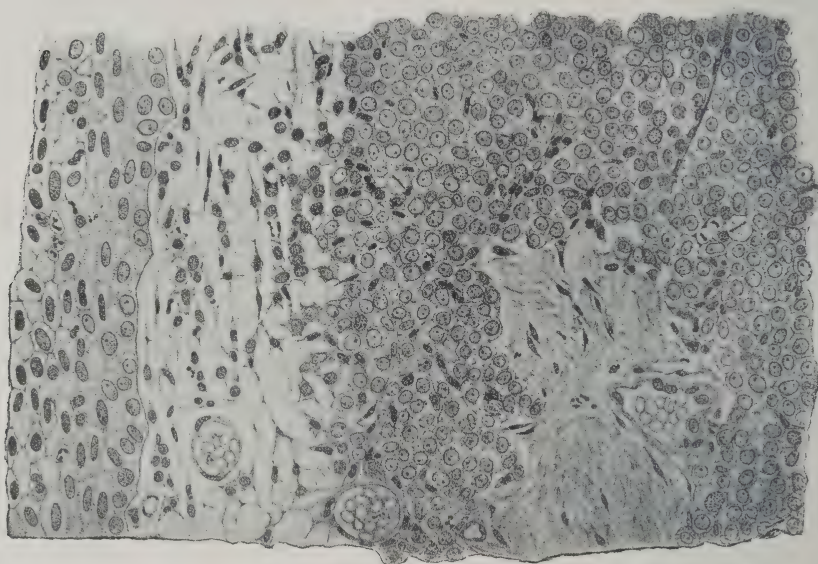

Fig. 125.—Infective Venereal Tumor of Vagina of Dog. Primary growth in vagina. Transformation of connective-tissue corpuscles into tumor cells. × 350. (*Bashford.*)

it closely resembles a malignant growth. He believes that its invariably infective history, the transformation of the surrounding connective-tissue corpuscles into tumor cells even in fully developed tumors, its artificial transmission, following the laws of such granulomata as tubercle or glanders, and the fact that it occurs naturally in animals before sexual maturity, all serve to distinguish it from true malignant tumors.

These objections, in the light of the facts already adduced, are not very convincing. On the other hand, we have shown that there is strong evidence of an infection in the primary sporadic tumors of the mouse, and we may add that many authorities concede that in primary tumors there is a transformation of normal epithelial cells into malignant epithelial cells at the margin of the tumor. Furthermore, the fact that the disease occurs naturally in young animals, which

is likewise true of sarcomata, should speak rather in favor of an analogy to malignant growths, which appear oftener in old age than otherwise. The one respect in which they appear to differ essentially from malignant tumors is that the cells do not appear to possess the power of limitless proliferation. It would seem that in transplantation experiments it would be a matter of great difficulty to determine whether or not all of the implanted cells disintegrate, and the essential point in which these tumors appear to differ from malignant tumors seems to be in the transformation of the normal cells of the host, in transplanted tumors, into tumor cells. The transplanted tumor in this case appears to repeat processes which are found in the development of sporadic tumors only, and as such it would appear that this tumor should in the future be the source of much fruitful investigation. The points which it has in common with true malignant tumors, the fact of its invariable infectivity, and the undoubted presence of an infective factor, should throw much light upon the much more elusive factors in malignant tumors.

Sticker, who has carried out extensive transplantation experiments with a tumor similar to the one described by Bashford, and who has carefully compared those transplanted by Smith and Washburn, Wehr (1888), and Geissler (1895), arrives at the conclusion that Bashford's interpretation of these tumors is not correct, and that they are genuine round-celled sarcomas. In this he is supported by Albrecht, Bollinger, Duerck, von Hansemann, Kitt, Luepke, Orth, Ribbert, Schmaus, Schmorl, Schuetz, Arnold, and Weigert, all of whom examined specimens of all five tumors (Smith and Washburn, Sticker, Wehr, Geissler, and Bashford), and diagnosed them to be typical round-celled sarcoma.

SUMMARY.

The following, then, are the arguments which have been adduced, from the modern research into cancer, in favor of the infectiousness of the process:

1. An analogy exists between certain of the changes in the epithelium in cancer and those occurring in the epithelium in certain of the acute exanthemata, notably variola and sheep-pox, known infectious diseases.

2. The almost exclusive appearance of cancer of the breast in elderly female mice which have been used extensively for breeding is best explained by the transference of some infective agent, through the medium of indiscriminate nursing, by offspring (Ehrlich).

3. Tumors in mice are almost never found alone. In breeding establishments, where one case appears it is always accompanied by others. Healthy mice, brought in contact with mice with primary tumors, acquire the same (Borrel).

4. The reappearance of sarcoma of the rat in a cage which had contained rats inoculated with sarcoma points to the possibility of cage infection in this form of cancer.

5. A gradual transformation of normal epithelial cells into cancer cells occurs at the margins of primary cancers (Orth).

6. The continued transplantation of mouse tumors increases rather than reduces their virulence. Certain mouse tumors under transplantation have acquired a virulence only comparable to that of an acute infectious process.

7. The transformation of an adenocarcinoma into a sarcoma (Ehrlich) is most easily explained by assuming the transference of an infective factor from the epithelium into the connective tissue of the stroma.

8. A certain number of mice are shown to possess a natural immunity which prevents inoculation with cancer. Spontaneous retrogression of cancer in mice is accompanied by histological appearances which show that the epithelium is not primarily injured, but that the stimulating factor is removed. Spontaneous retrogression is accompanied by a type of acquired immunity which prevents the successful reinoculation of the animal, and under favorable conditions this factor appears to be present in the blood and behaves not unlike the known antitoxins to infectious processes.

9. The blood of spontaneously recovered mice, when added to cancer material before transplantation, removes from it the power of continued proliferation. There is no evidence of cytolytic action (Clowes).

10. Tumors retrograding under the influence of the x-ray and radium present exactly the histological picture of tumors spontaneously retrograding. The stimulating factor seems to be removed from the epithelium through the aid of the immune mechanism.

11. The epithelial cells of the deeper layers of warts, after successful treatment with the x-ray, no longer proliferate to form a new wart, but reproduce normal skin (Perthes), showing that the stimulus to proliferation has been removed and that there remain epithelial cells capable of normal proliferating function.

12. The unknown factor in cancer is apparently added to normal epithelium, from which it can be removed, leaving normal epithelium. Through the proliferation of the cells of the cancer, which increase enormously, this factor must of necessity gradually increase in amount. The increase in bulk, through transplantation in mouse tumors, is associated with increased virulence. The only known agent which can fulfil these conditions is a living organism. The unknown factor may be an ultramicroscopic organism, or one that is simply undemonstrable. Filtration experiments in infectious diseases of unknown etiology are not competent to throw any light on this phase of the subject.

13. Infectious venereal granuloma of the dog, an undoubtedly infectious tumor, presents certain points of similarity to malignant processes. The tumor grows largely through karyokinesis of the tumor cells which are derived from the connective-tissue cells of the host (Bashford). The cells do not appear to possess the power of limitless proliferation, although perhaps this is not conclusively proven.

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PART II.

COMPLICATIONS AND SEQUELÆ.

INFECTIONS WHICH SOMETIMES OCCUR IN VARIOUS SURGICAL DISEASES AND CONDITIONS.

By PAUL MONROE PILCHER, M.D., New York.

WOUND INFECTIONS.

WITHOUT exception, surgical infection is due to bacterial activity. Bacteria however, may be present in a wound without giving rise to inflammation. The virulence of the micro-organisms, the environment in which they find themselves, the ability of the tissues to protect themselves—these and many other factors influence the course of the infection.

A freshly made wound, presenting crushed and partly devitalized tissue, and a cavity filled with coagulated blood and serum, furnish the ideal conditions for the development of an infective inflammation.

It is generally accepted that the Staphylococcus pyogenes aureus and albus and the Streptococcus pyogenes, separately or associated, are in most cases the infective agents. Many other bacteria, however, may be the etiological factors. Among the most important of these are the Bacillus coli communis, Bacillus pyocyaneus, Proteus vulgaris, Micrococcus tetragenus, Bacillus of Friedlander, Bacillus typhosus, the Gonococcus, Streptococcus erysipelatis, and the Pneumococcus. Some of these bacteria, under ordinary conditions, do not produce a suppurative inflammation; on the contrary, they are more frequently associated with some other form of exudation, but under special circumstances they may produce suppuration.

The presence of bacterial activity in a wound gives rise to inflammation, clinically recognized by the local redness, swelling, heat, and pain, and the occurrence of fever. These phenomena must necessarily vary according to the character of the tissue which is the seat of the inflammation. Redness would necessarily be absent in a non-vascular tissue, while heat as a sign of inflammation has not been observed in many of the viscera. The degree of swelling and pain varies with the tissue and the individual attacked.

These signs are due to pathological changes in the vessels and tissues. At first there is an active dilatation of all of the vessels and an increase in the rapidity of the flow of blood through the tissues; in other words, a *hyperæmia.* This gives rise to the redness and heat. Following this there are a passive dilatation of the capillaries and veins and a gradual slowing of the blood. The relative number of white blood cells is increased, more especially in the veins. Gradually,

more or less complete stasis of the blood current takes place, and the white blood corpuscles migrate through the walls of the veins and capillaries, completely surrounding the walls of the vessels, and pass on out into the contiguous tissue. In the more severe forms there is also a diapedesis of the red blood cells, which is held to be a passive transudation. The swelling is due to the fluid which is exuded from the vessels into the lymph spaces and then into the tissue itself. In inflammation of the peritoneum or pleura the exudate passes directly into the peritoneal or pleural cavities. This exudate varies greatly under different conditions. It may be a purely serous exudate, but more frequently it is a sero-fibrinous exudate. According to Councilman, the fibrin is formed entirely outside of the vessels. The fibrinogen contained in the serous exudate is converted into fibrin in the presence of a ferment produced by the degenerated cells. The exudate in the case of a suppurative inflammation is characterized by the presence of a certain number of degenerated leucocytes, an increase in the amount of albumin, and a greater degree of coagulability (Hildebrand). It is especially rich in cells. This form of exudate is frequently met with on the surface of wounds, and is known as a *fibrinous* or *croupous exudate.* It is generally associated with superficial necrosis. Again, in suppurative inflammations the production of pus cells may greatly predominate, and small cavities be formed, containing the pus cells, exudate, and necrotic tissue, and thus an *abscess* will be formed. In such cases the amount of fibrin found is generally relatively small. According to Councilman, the immediate effect of the presence of growing bacteria in a wound is the production of an area of necrosis around them. Around this necrotic area or even within it are seen many leucocytes of the polymorphonuclear variety. They form a definite wall. The chemotactic properties of the necrotic tissue and the bacterial products increase, and the leucocytes invade the necrotic mass. The central mass liquefies, becomes circumscribed by granulation tissue, and an abscess is formed, the liquid contents of which are known as *pus.* When the infection is more severe there is often a diapedesis or even a true exudation of red blood cells, and we have the *hemorrhagic exudate,* which almost always signifies a necrotic process. Necrosis always accompanies a suppurative inflammation in the tissues, and consequently there is a loss of tissue. If this is superficial it is seen as an *ulcer;* or, if confined within the tissues, it forms an *abscess.* Always at the periphery of a suppurative inflammation granulation tissue is built up.

Suppuration may be confined to the surface of the wound, or, beginning in the wound, it may spread rapidly and involve not only the adjacent tissues, but also the general system.

As has been already stated, the cause of suppuration in a wound is bacterial activity. Generally, the bacteria gain entrance by direct inoculation of the wound. There are, however, other avenues of entrance. It has been conclusively demonstrated that under certain conditions bacteria may pass through

the epithelium covering the tonsils and gain entrance to the general circulation. This is also true of the mucous membranes lining the intestinal and respiratory tracts. These facts are of special interest in explaining those cases of infection in which there has been no discoverable external wound or those in which an injury, such as a simple fracture, becomes infected and gives rise to an extensive suppurative process.

It must be remembered that not all bacteria which gain access to the wound bring about suppuration. In the first place, they must find a proper medium for their activities and development. It is true, too, of some bacteria that they cause suppuration in one animal and not in another; also some bacteria acting alone are non-pathogenic, but when associated with other forms of bacteria they become actively pathogenic. Having gained access to the wound, they may actively proliferate and invade the surrounding tissues or pass directly into the lymph or blood streams and bring about a general infection.

The next question of importance is, *How do the bacteria cause injury to the tissues?* As a result of exhaustive researches, the conclusion has been reached that the phenomenon is fundamentally a chemical process. The bacteria themselves secrete ferments which act directly upon the tissues, exerting a peculiar digestive action upon them. They further assimilate certain substances from the surrounding tissues and excrete others. These latter assimilation products are chiefly ptomaïns, such as putrescin, sepsin, and cadaverin, and are poisonous; so also are the bacterial proteins and toxalbumins (Hildebrand). These poisons act locally upon the tissues, causing necrosis, and the combined resorption into the system of the products of decomposition and the bacterial toxins gives rise to the constitutional intoxication.

Of all the pus-producing bacteria, the Staphylococcus pyogenes aureus and albus and the Streptococcus pyogenes are the most frequently met with. The staphylococcus is most often the cause of localized suppurative processes, such as furunculosis, carbuncles, localized abscess, acute osteomyelitis and periostitis, pustular skin diseases, empyema, etc. The streptococci are seldom found in these conditions, but are found more frequently in the phlegmonous inflammations. When these cocci—either separately or in combination—are introduced into a wound in sufficient numbers, they give rise to a suppurative inflammation. The local tissue which is infected helps to limit the disease by building up granulation tissue. The vascular system supplies the fibrinogen and the leucocytes. The leucocytes invade the inflammatory tissue, and, acting as phagocytes, help to limit the growth of the cocci and eventually to destroy them. It is questioned by many whether the leucocytes act as true phagocytes; be this as it may, they certainly play an active part in inhibiting the spread of the cocci. There are also in the blood a number of other substances, which are directly antagonistic to the bacteria and their products. Two of these, agglutinin and bacteriolysin, are produced in the spleen, bone-marrow, and lymph nodes,

and seem to act by paralyzing the bacteria and preparing them for the attack of the alexins. Alexin is produced in the blood itself, and is deadly to the cocci. There is also present in the blood serum a much more potent factor, viz., antitoxin, which is a true bactericide.

The protective forces of the body may temporarily limit the advance of a suppurative inflammation without entirely stamping it out. An example of this may be occasionally found in bone abscesses, which subside after a period of considerable activity, remain quiescent for years, and then suddenly give rise to severe symptoms. The infectious agents in such cases are almost always staphylococci.

Another micro-organism of frequent occurrence in suppurative processes is the Bacillus pyocyaneus. Many authors maintain that it exists simply as a saprophyte on the skin, and that, acting alone, it does not cause suppuration. Other writers, however, believe that under certain conditions it must be classed as a pyogenic bacterium. We do know that, when associated with streptococcic or staphylococcic infections, it becomes active and gives rise to a peculiar exudate, called *green* or *blue pus*. It shows its blue color only in the presence of oxygen.

When pyogenic bacteria become active in the tissues of the body, various types of inflammation may follow. If the process is confined within the tissues an abscess results, which may spread or remain localized. If a general infiltration of the tissues takes place, it is known as a *phlegmonous inflammation*. When the abscess is superficial and opens on the skin, it is called thereafter an *ulcer*. In such a condition there is always a loss of substance, exposing the deeper tissues. Certain localized deep-seated inflammations of the skin are termed *furuncles* or *boils*. Cellulitis of the soft parts of the fingers or toes are called *felons*, or *paronychiæ*.

In all of these conditions the ordinary phenomena of inflammation are evident in varying degrees. The constitutional symptoms are sometimes marked, but they rapidly subside upon removal of the local cause.

Infection complicating the healing of a wound may manifest itself in various ways. It may cause a simple inflammation and active suppuration may not occur, or a most active and virulent suppurative or gangrenous inflammation may result. The process may be limited to the wound and its immediate neighborhood, or it may be progressive and involve large areas.

SIMPLE INFECTION.

When a recent wound is infected, it may not show signs of inflammation until the second or third day. If the wound has been sutured, the constitutional symptoms may be the first evidence of the infection. The patient complains of headache or a feeling of general malaise. There may be anorexia or even nausea. Unless the infection is extensive, vomiting does not generally occur. The most constant symptom is *fever*. At first there is little to differentiate this fever from

the ordinary aseptic wound fever which almost always is noticed in the healing of extensive wounds, especially where there is much loss of blood or destruction of tissue. Such fevers usually subside on the second or third day following the injury to the tissues. The fever accompanying an infection, however, does not subside. At first the body temperature may not be very high, but gradually it increases, until on the third or fourth day it may register 103° F. or higher. The pulse varies. Usually there is a corresponding increase, but at first it may not be marked. An examination of the wound, if it be sutured, will show a redness and œdema along the suture line. If the infection be deep-seated there may be no superficial signs, but palpation will demonstrate induration and tenderness. The patient may complain of local pain. When the infection is extensive all the symptoms of a suppurative inflammation are present. If the wound is an open one there will be noted an increase in the amount of the secretion which it furnishes. At first, this secretion is serosanguineous, but later it becomes purulent. The edges of the wound become swollen and œdematous, and, as the infection spreads into the connective tissue and between the muscular septa, the usual symptoms of a phlegmonous inflammation develop. If the infected area is not incised or opened it may become localized, being circumscribed by granulation tissue—in other words, an abscess may form; or the infection may rapidly spread and give rise to an extensive phlegmonous inflammation. Again, the toxins and the microorganisms may be rapidly absorbed and give rise to septicæmia or pyæmia. Usually, when the process is discovered early and free drainage is provided, combined with the proper antiseptic treatment of the wound, the signs and symptoms of inflammation gradually subside and the wound heals by granulation. If the superficial portions of the wound heal first, retention of the secretions and purulent exudate may occur, and then the constitutional symptoms due to the absorption of the toxins will again appear. During the course of healing of such infective processes, if the infection be deep-seated, *sinuses* and *fistulæ* often result, and these do not heal until all of the necrotic or infected tissue has come away.

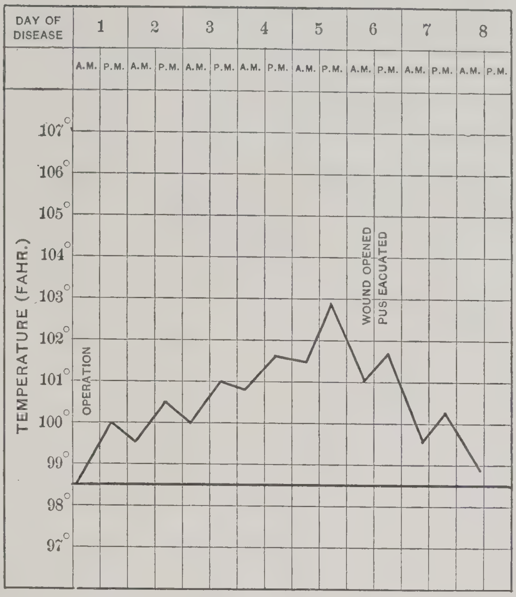


FIG. 126.—Temperature Chart of a Case of Infected Wound Following Operation.

ACUTE SEPTIC PHLEGMONA.

Aside from the simple wound infections and suppurative phlegmona which tend to remain more or less localized or are easily controlled by treatment, there are a number of acute infective processes which originate in a wound and rapidly and progressively spread, often giving rise to most alarming symptoms. The mildest form of this type of infection is the so-called *progressive phlegmonous infiltration,* which spreads rapidly from the seat of the original infection, involving the connective tissue, the intermuscular septa, the fascia, and the tendon sheaths, without leading to localized pus formation. It is most frequently seen in compound fractures of the extremities or in extensive crushing injuries. The first symptoms usually appear within three or four days after the injury. There is a

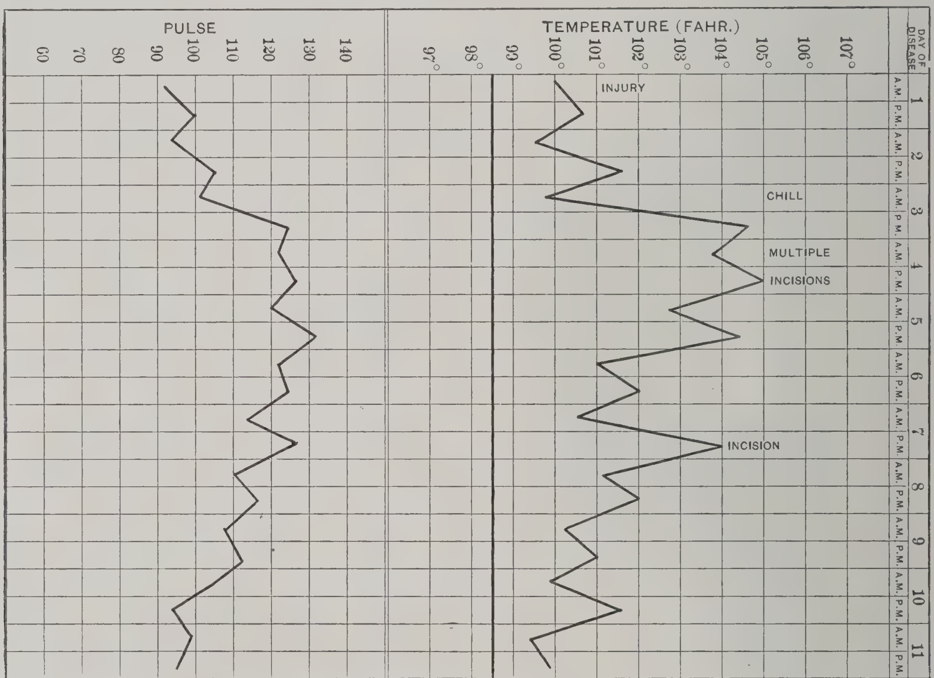


FIG. 127.—Temperature and Pulse Curves of a Case of Acute Septic Phlegmon following Extensive Injury of Foot.

chill, accompanied by a rapid rise in temperature and a rapid pulse. There is a foul-smelling discharge from the wound and a rapidly extending œdema. The tissues are infiltrated with a foul-smelling sero-purulent exudate, which, if not relieved, results in extensive necrosis and diffuse suppuration. The constitutional symptoms are marked.

ACUTE PURULENT ŒDEMA.

Another form of the same type of infective processes is the *acute purulent œdema,* first described by Pirogoff. The original wound may have been slight, or

it may follow or complicate extensive crushing injuries. The progress of the infection is rapid and virulent. Within from twelve to twenty-four hours after the injury the part, usually an extremity, becomes rapidly swollen and œdematous. The sero-sanguineous discharge from the wound becomes sero-purulent and is very offensive. Marked constitutional symptoms arise. The body temperature is high and the pulse rate rapid. The swelling rapidly increases. If the tissues are incised they will be found to be everywhere infiltrated with cloudy fluid. Portions of the tissues are already necrotic, despite the short duration of the disease. The odor of the secretions is most foul. The lymphatics are extensively involved. In a few days multiple suppurative foci develop throughout the tissues. The abscess cavities are filled with offensive pus, necrotic tissue, and frequently foul-smelling gas. Often an entire extremity is involved in the process. The disease generally terminates in a fatal septicæmia.

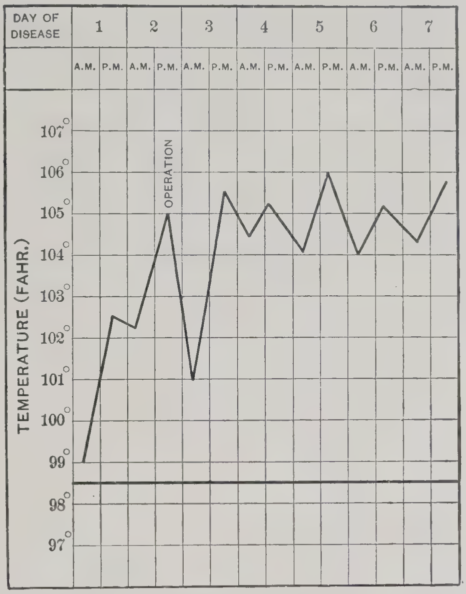


FIG. 128.—Temperature Chart of a Case of Acute Purulent Œdema, which Terminated Fatally.

Gangrène Foudroyante.

The *gangrène foudroyante,* first described by Maisonneuve, is closely allied to the process which has just been described. It most frequently follows a bone injury, and has often been known to follow a crushing injury of the foot or leg. It is characterized by the progressive character of the infection, the rapid course, the gangrenous destruction of the tissues, and the production of gas abscesses and a spreading emphysema (Hildebrand). The cause of the infection is the Bacillus of malignant œdema (Koch).

In such cases the extremity swells rapidly and soon gives evidence of a spreading emphysema. The secretion from the wound is sero-sanguineous and scanty. The extremity shows an advancing dusky œdema. In twenty-four hours the entire limb may be involved. The skin crackles when touched (Spencer). The veins appear as bluish stripes on the brownish-red skin. The constitutional symptoms vary. The body temperature may not be high or we may have a typical septic curve. Frequently diarrhœa and involuntary evacuations of the bladder and rectum occur. The patient is restless. The pulse is rapid. Commencing gangrene is seen. Incisions show multiple abscesses containing pus and foul-

smelling gas. The gangrene extends rapidly. The body temperature gradually falls, sometimes becoming subnormal, and death follows.

The prognosis is bad. Death generally occurs, but cases of recovery have been reported. The treatment consists of multiple incisions and continuous irrigations or early amputation..

Lymphangitis.

Infections may spread from the original focus and involve the lymph channels, and an endolymphangitis or perilymphangitis be set up. It may further reach the lymph nodes and cause a lymphadenitis. The consideration of this form of infection will be taken up in the article devoted to the Diseases of the Lymphatics.

Local Infections of Granulating Wounds.

There is a certain form of infection which attacks not only recent wounds, but also wounds which are already covered with healthy granulations. This disease is commonly known as *hospital gangrene.* The etiology as yet is uncertain. Numerous micro-organisms have been found, but no particular one has been isolated which is known to bring about the disease. The first manifestations are a progressive infiltration and a coagulation necrosis of the granulations, the process spreading rapidly at the periphery of the ulcer and at the same time penetrating deeply into the tissues. The disease is further characterized by a gangrenous destruction of the inflamed tissue. The first symptoms are entirely local, and the disease spreads by attacking the contiguous tissues.

Three forms have been described: (1) The croupous or diphtheritic; (2) the ulcerating; (3) the pulpous form. The first is characterized by the formation of a pseudo-membrane on the surface of the granulations, underneath which extensive necrosis and gangrene of the tissues rapidly develop. The surrounding tissues are not much inflamed. The second form is characterized by a rapidly spreading ulcer, with necrosis of the underlying tissues and a copious, foul-smelling discharge. The third form is the most virulent. There is a rapid puffing up or swelling of the tissues. Hemorrhages take place within the granulations, and they undergo a purulent necrosis, followed by gangrene and a separation of the entire mass. The surrounding tissues are markedly œdematous and inflamed, and the ulcerated surfaces are exquisitely tender. In all of these forms the process spreads rapidly, attacking and destroying everything in its path.

The constitutional symptoms are marked, and present all the phenomena of a general systemic intoxication.

The prognosis is grave. It naturally varies with the form of the disease, the pulpous form being the most fatal. In the Civil War in this country the mortality was 45.6 per cent.

SEPTICÆMIA.

The term septicæmia is no longer accepted in the sense of its literal translation, but nevertheless, on account of long usage and general acceptance, it is still so employed. It is not possible to define it pathologically, because its limits are not fixed. We accept it more as a word which is suitable for designating the degree or the severity of certain general intoxications and infections. Clinically we speak of an infection as local when the predominant symptoms are due to the local disturbance, the systemic manifestations appearing as secondary. In the case of an abscess which is confined, the surrounding walls exert a certain amount of pressure upon the contained pus, and resorption of the toxic materials takes place. Frequently bacteria as well are found in the blood, and we practically have a septicæmia. When the abscess is opened and the tension is relieved, the general symptoms subside and the infection is then merely local. If, however, upon evacuation of the pus, the general symptoms continue and we have a systemic intoxication, we speak of it as a septicæmia or a general septic infection.

Gussenbauer has defined septicæmia as a "general disease of the body, which results from the introduction into the circulation of the products of decomposition, and which is characterized by definite changes in the blood, a typical succession of inflammatory processes, and a continuous fever, together with peculiar nervous symptoms and critical discharges." The extensive researches of Ogsten, Rosenbach, Doyen, von Eiselsberg, and others have taught us that the general systemic disease known as septicæmia depends upon the introduction of pathogenic, especially pyogenic, micro-organisms into the general circulation. However, there is another general intoxication, known as sapræmia, or septic intoxication. This intoxication, which results from the absorption of the products of putrefaction, is so closely allied clinically to true septicæmia that it must be considered in connection with it.

Sapræmia is a septic intoxication or toxæmia, due to the absorption of toxins formed by the bacteria of putrefaction. It should not be confounded with the so-called aseptic wound fever, which results from the absorption of the products of aseptic tissue necrosis, and which gives rise to a systemic intoxication. In sapræmia we have a definite pathological lesion; that is, the infection of necrotic tissue with putrefactive bacteria. Among the most important of these micro-organisms may be mentioned the Proteus vulgaris. Locally, as a result of the putrefactive processes, certain ptomaïns are elaborated, which are absorbed and bring about a general septic intoxication.

Symptoms and Diagnosis.—The symptoms which develop are those of a local putrefactive process combined with the constitutional symptoms of a ptomaïn poisoning which is gradually progressive, which acts as a depressant on the nervous system, and which gives rise to considerable fever. The local focus of in-

fection is generally unmistakable. Frequently the interior of the uterus is the seat of the disease. Following childbirth, there is an infection of the secundines retained within the uterus; the foul discharge and the febrile movement direct our attention to the condition, and the diagnosis is then easy. More frequently, large masses of gangrenous or sloughing tissue in a wound undergo putrefaction, and the foul odor of the putrefying tissues, as well as the visual picture, establishes the diagnosis. The constitutional symptoms are seldom initiated by an actual chill, but more frequently the patient complains of a headache, loss of appetite and general malaise. While there may not be an actual chill there is usually a sensation of chilliness. At first the body temperature rises to 99.5° or 100° F. The following morning it may again be normal. On the afternoon of the second day the temperature becomes higher, and we have a

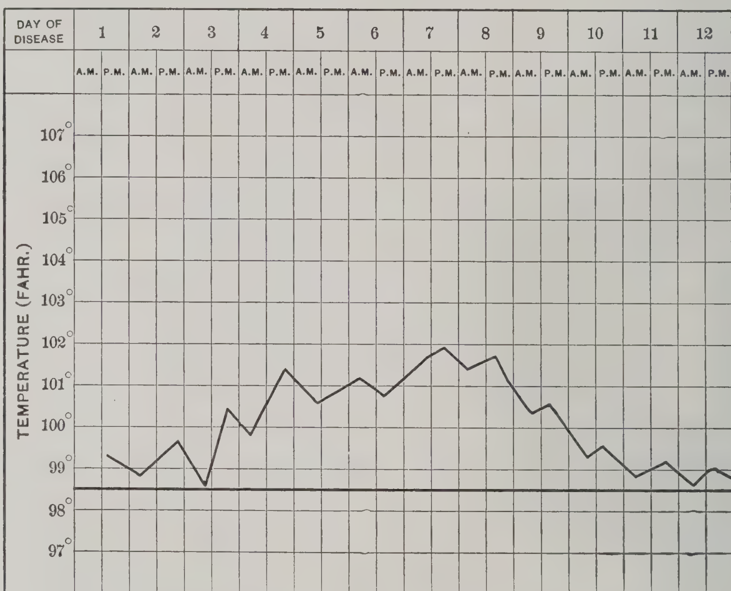


FIG. 129.—Temperature Curve of a Case of a Mild Grade of Sapræmia.

continuous fever with slight remissions, its severity being directly proportionate to the extent of the local putrefactive process. If the diseased tissue is not removed, the headache becomes more intense, the body temperature rises, vomiting and diarrhœa occur. An examination of the blood will show degenerative changes: in the more severe cases poikylocytosis and diminution in the number of red blood cells, and a moderate leucocytosis. The pulse, at first soft and compressible, becomes rapid and weak. The tongue is furred and dry. The urine is scanty. Gradually the poison overcomes the nervous system, delirium follows restlessness, and coma develops. Micturition and defecation become involuntary. The pupils become dilated, the patient is covered with a cold, clammy perspiration, the pulse becomes irregular and feeble, and death occurs. In some

cases the disease is marked by intense gastro-intestinal symptoms. The vomiting and purging may be so severe that the case may simulate cholera. In the milder cases, in which the amount of tissue acted upon by the saprophytes is small, the disease soon runs its course and subsides.

Prognosis.—In uncomplicated cases the prognosis is good, because the disease is easily recognized, and prompt treatment is usually followed by a rapid subsidence of the symptoms. The great danger lies in the possibility of secondary infection, which, when it occurs, generally gives rise to a severe form of septic infection.

Treatment.—Prophylactic measures in this condition are of paramount importance; they comprise the removal, wherever possible, of all necrotic tissue which is liable to undergo putrefaction, or, if this be not possible, the sterilization (through chemical means) of the necrotic tissue and the prevention of infection. When the disease is established, prompt measures must be taken to remove all of the infected material and to prevent a reaccumulation. The general systemic treatment will be discussed under the treatment of septicæmia in general.

Septicæmia.—Under this head we will consider that septic infection of the entire body which is brought about by various kinds of bacteria, and which gives rise to the symptoms of a constitutional intoxication without the clinical signs of metastases. This includes the various forms of septicæmia designated as toxæmia, toxinæmia, pyotoxinæmia, bacteriæmia, and pyosepticæmia.

Etiology.—What has been said concerning the relation of bacteria to the suppurative inflammations is also applicable to septicæmia. There is no specific micro-organism. Unquestionably the staphylococci and the streptococci play the most important rôle. We may have more than one variety of bacteria present in the same case, as in double infection, or we may have a secondary infection. It is still doubtful whether the bacteria which gain entrance to the general circulation increase and produce their toxins in the blood. Brunner holds that an acute mycosis never is met with in the human blood, and he believes that there never occurs any marked growth of bacteria in the blood. He further maintains that the micro-organisms are prone to collect in the parenchymatous organs, and that, in the acute cases, they set up metastatic processes, which, however, remain microscopically small, the duration of the disease being too short to develop macroscopic foci or to manifest itself by any clinical evidence. The majority of investigators believe, however, that the bacteria, after gaining entrance to the blood, increase and elaborate their poisons in the blood, and then, independently of any other suppurative foci, may cause death. In certain severe local infections it has already been noted that the staphylococci and streptococci produce very poisonous toxoproteins and toxalbumins, and that these poisons may be reabsorbed and give rise to septicæmia. It has also been shown that in such cases numerous micro-organisms reach the circu-

lation, but the action of the reabsorbed toxins is so rapid and severe that they produce the symptoms of the disease before the micro-organisms have had time to increase and become active. This form of sepsis is called *toxinæmia.* In other cases the bacteria themselves rapidly reach the circulation, and there increase and produce toxins, and we have a *bacteriæmia.*

The question naturally arises, How do the toxins and the bacteria gain entrance to the blood? In the rapidly fatal cases it seems most probable that they pass directly into the lymph spaces and are in this manner thrown into the general circulation. In other cases, again, they must first penetrate the granulation tissue, and, passing along the main lymph channels and overcoming the resistance of the lymph nodes, enter the blood.

Symptoms.—The different forms of septicæmia differ so widely in their clinical manifestations that it will be best first to consider the symptoms in general, and then to present some of the more important types of the disease.

The symptom to which our attention is first called in septicæmia is *fever.* In general, it shows itself at first as a moderately high, continuous fever. The morning and evening temperatures, as a rule, vary but little. Sometimes, but not always, the fever is ushered in by a chill or a feeling of chilliness. In a pure septicæmia repeated chills seldom occur. In the more severe types of the disease, especially when due to a mixed infection and when there is present a large amount of necrotic and purulent material in the wound, the body temperature is high. In other cases the temperature may be low and even subnormal, and this is always an unfavorable sign, especially when accompanied by a rapid and feeble pulse. When convalescence is established the body temperature gradually sinks to normal.

The pulse is a much more important criterion of the patient's condition than the temperature. At first, in the milder forms, the heart's action is not especially accelerated, but as the disease progresses the arterial tension is lowered and the pulse becomes rapid and feeble. In the most virulent forms the heart's action quickly loses in power, and many circulatory disturbances make their appearance.

The nervous system is very soon affected by the toxins. At first the patient may complain of headache and a feeling of general discomfort or pain in the wound, but this soon gives way to apathy and lack of interest in his condition and his surroundings. This state may alternate with restlessness, but gradually stupor comes on, and in the fatal cases coma and death follow. Delirium does not generally occur.

A great change takes place in the patient's general condition. There is *profound prostration.* The surface of the body, at first dry and hot, later is bathed in perspiration, the skin feeling cold and cadaveric. The patient loses weight rapidly. The expression is listless, the face being drawn and colorless; the eyes are sunken, and the alæ nasi dilated. The tongue at first is thickly coated, and later becomes covered with dry, hard crusts.

Almost always the patients suffer from severe gastro-intestinal symptoms. At first, there is loss of appetite and the thirst increases. Nausea and vomiting are frequently observed, and diarrhœa is the rule. In the more severe cases there may be active vomiting and purging, as in cholera.

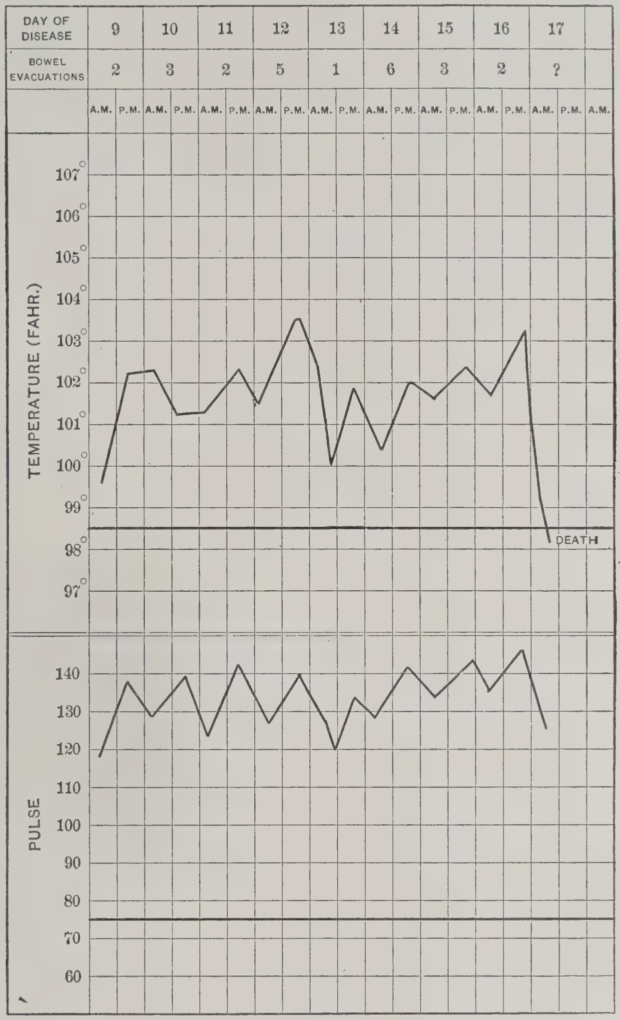


FIG. 130.—Typical Temperature and Pulse Chart of Second Week of Septicæmia. Note rapidity of pulse and frequent evacuations of bowels.

The skin often shows a yellowish tinge, and a variety of eruptions may appear.

The urine shows albumin and casts.

If the disease responds to treatment, a general improvement of the sensorium is first noted; the pulse becomes a little stronger, although still rapid; the body temperature gradually subsides, often showing at first marked morning remissions, until finally the evening rise disappears; the desire for food gradually returns; the heart is the last entirely to recover its normal condition.

As has been already stated, we recognize clinically several different types of septicæmia.

Type I.

The patient has suffered a compound fracture of one of the long bones. A few days later the signs of infection develop in the wound. Local treatment does not dimin sh the inflammation, and active suppuration takes place. This rapidly spreads and the lymph channels become involved, and all the symptoms of a severe general septic intoxication develop. There is no attempt at healing in the wound. The body temperature has become continuously high and the pulse increasingly rapid. As the disease progresses the patient becomes apathetic. Vomiting occurs. There are four or five loose diarrhœal movements of the bowels. Examination of the blood may demonstrate the presence of pyogenic bacteria. Prompt and extensive local incisions and antiseptic applications may check the further advance of the disease. The constitutional symptoms gradually disappear, convalescence is established, and the wound heals.

Or the disease may be much longer in its course and not react so quickly to treatment. The following case will serve as an example:

A young and healthy adult receives a gunshot wound of the shoulder. He is taken to the hospital and the wound is treated antiseptically. At first there are symptoms of shock, but these rapidly pass away and the temperature and pulse are normal. An examination of the wound shows the presence of dark fluid blood and serum, which are easily expressed from the cleanly cut opening of the bullet wound. Surrounding this opening there is more or less œdematous swelling of the parts. Pressure elicits pain. Second day: The general condition is satisfactory. The body temperature is 100° F.; pulse, of good quality and about 100 to the minute. Pain only on muscular motion. Third day: Patient feels ill, is very thirsty, and has no appetite. Evening temperature is 103°; pulse, 110. Some pain in shoulder, increased by motion. Fourth day: The dressings are changed; they are found to be dry. The wound is covered with a dry, hæmato-fibrinous exudate; no pus; no symptoms of local infection. Evening temperature, 102.5°; pulse, 105. Tongue coated and moist. Fifth day: Sleeps poorly. Great pain in shoulder, increasing thirst. Evening temperature, 103.6°; pulse, 120 and of good quality. Sixth day: Morning temperature, 102°; pulse, 110. The pain in the shoulder has increased. The tongue is dry and coated. The patient is restless. On inspection the wound shows the entrance point closed by a dry, hard crust. The surrounding tissues, however, are swollen for some distance from the wound, moderately hyperæmic, and very painful to pressure. It is possible to express some pus from the wound. Immediately the region is incised and the entire course of the bullet is laid open. Considerable purulent exudate is discovered, and one or two small abscesses are opened. The tissues are discolored, œdematous, and infiltrated. Some bone destruction is found. The purulent exudate is seen to issue from fissures in the surrounding

tissue. Some bone splinters, possibly a piece of clothing, and the bullet are removed. The wound is treated antiseptically and drained. Seventh day: General condition somewhat improved, less pain. Evening temperature, 102°; pulse, 110 and of good quality. During the next five days the purulent exudate in the wound becomes progressively less. Granulations begin to appear. The morning and evening temperatures are lower. On the following day, however, the patient complains again of severe pain in the shoulder. Evening tempera-

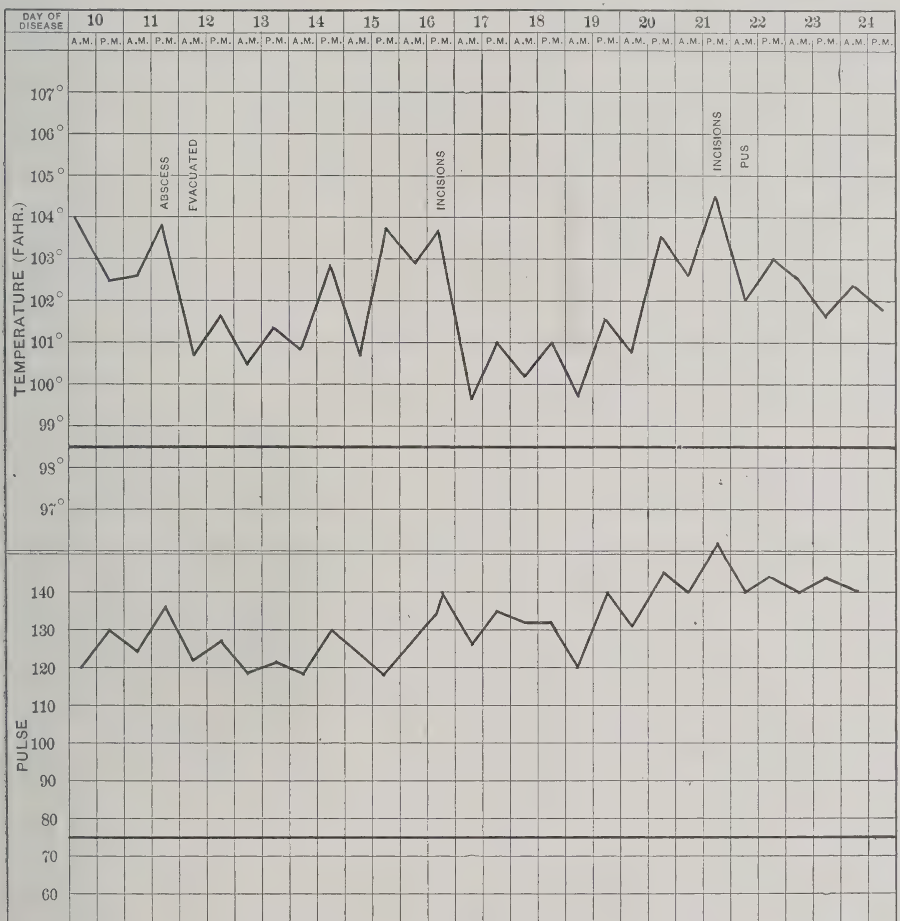


FIG. 131.—Temperature and Pulse Curves of a Case of Pyosepticæmia. Formation of multiple pus-containing cavities which spread rapidly. In some places extensive areas of necrotic, foul-smelling tissue were found; also gas abscesses. Multiple and repeated incisions.

ture jumps up to 103°; pulse, 120. Considerable prostration. Patient listless. Exploration of the wound does not demonstrate any new pus foci; simply discolored, œdematous, infiltrated tissues. Despite wide incisions, the evening temperature reaches 103.5°; pulse, 130 and irregular. Energetic surgical treatment fails to produce any change for the better in the patient's condition. The average evening temperature during the next week is about 102.5°, with morning remissions to 99.5° or 100°; the pulse between 90 and 120. At the end of

the second week staphylococci are found in the blood and urine. At about the same time a retained collection of pus is found in the wound. There is no odor of decomposition or putrefaction. Occasionally the body temperature shoots up to 103° or even 104°, the pulse to 120. At the end of three weeks the temperature remains the same, but the pulse, on the slightest exertion, runs up to 150 and becomes quite irregular. Small necrotic areas are found burrowing outward from the wound. Multiple incisions are made. Still the temperature remains high, now being almost continuously above 103° and reaching as high as 104.5°. Pulse, 140 to 150, and of very poor quality. Patient sleeping most of the time. Delirious at times. Repeated blood cultures show the presence of the Staphylococcus pyogenes in the blood. All of this time the wound has been most energetically treated, and the most advanced therapeutic measures have been used to sustain the strength of the patient and overcome the poisons of the disease. Finally, at the end of the fourth or fifth week, the wound begins to look healthier. The temperature does not rise so high in the evening and the pulse is more regular. Gradually the constitutional symptoms abate, the local conditions improve, and at the end of the eighth or ninth week the temperature remains normal. The pulse is still rapid, 110 to 120, but it gradually returns to normal.

Into this class of septicæmia fall those cases in which there is no mixed infection and where the symptoms are due more to the toxins and ferments of the bacteria themselves than to the added resorption of the products of decomposition. Of course necrosis always occurs in such cases, but it is not by any means a prominent symptom. In this class of cases the onset often is gradual, but it may be sudden and severe. Many examples of these cases are seen in infections of the knee-joint. The area of local tissue-necrosis is not great, but the bacteria and their toxins are rapidly reabsorbed and give rise to grave constitutional symptoms. Another example is seen in the so-called post-mortem infections. Here there is seldom much tissue-necrosis, and often the local infection is so insignificant that it entirely escapes the notice of the surgeon. The development of the general infection, or bacteriæmia, is rapid and severe, and the disease often proves fatal in a remarkably short time.

The prognosis in this type of cases depends upon the early recognition of the primary focus of infection and upon the thoroughness and promptness with which the antiseptic treatment is carried out. Even after the bacteria have reached the general circulation, if the local focus of infection can be entirely eradicated, the chances are that the bacteriæmia will rapidly disappear

Type II.

This type comprises those cases which are known as *cryptogenetic septicæmia.* In the one set of cases there is a history of injury. A simple fracture, a contused wound or hæmatoma without external wound, or a crushing injury of a

bone are among the most common examples. Following these injuries are the usual signs of traumatic inflammation. This inflammation does not undergo resolution, but very soon gives rise to a decided febrile movement, which may be initiated by a chill or sensation of chilliness. All the local signs of a suppurative inflammation appear. The constitutional symptoms increase and a septicæmia develops.

In the other set of cases there is no history of any wound or injury, and without known cause the patients gradually or suddenly develop the symptoms of a general septic infection. The course may be acute, subacute, or chronic, but as a rule the symptoms of multiple pus foci develop, and we have a transition into pyæmia.

The etiology of these conditions has already been discussed in the article on Inflammation.

Type III.

To this type belong the cases of septicæmia due to a mixed infection.

Under this division of septicæmia we have to deal with a condition whose manifestations vary according to the local pathological conditions. It is brought about by a number of different micro-organisms working together, a poly-infection, causing extensive local necrosis and decomposition, as well as a general infection of the system. Often streptococci, the Bacillus coli communis, the Proteus vulgaris, and the Bacillus pyocyaneus are associated together in such a process. The symptoms naturally vary, but an example, taken from actual experience, may best be used to illustrate the condition.

The patient has received some injury to the spine, and as a result a chronic myelitis has been set up. Despite careful and constant attention, the patient's condition grows worse and a number of bed-sores develop. One or more of them grow larger and become infected. The skin is undermined and a collection of foul-smelling pus is evacuated. The patient has developed a high fever, with all the symptoms of a general septic intoxication. A large fluctuating abscess forms on the thigh and spreads rapidly to the knee. When it is opened, a large quantity of foul, gas-containing pus escapes. Large masses of necrotic tissue are seen everywhere. There is no appearance of healthy granulation tissue. An examination of the urine shows Bacterium coli. The blood shows a pure culture of Proteus vulgaris. The evacuated pus contains cultures of Bacterium coli, Proteus vulgaris, and streptococci. These agents working together rapidly overcome the patient, who dies in coma.

The symptoms, then, are those of an extensive local necrosis combined with the symptoms of a general septic infection. The body temperature is generally high, but very irregular and remittent in type. The pulse rate is high and the nervous system is markedly involved. The blood changes are not constant, and it is not always possible to demonstrate bacteria in the blood. As a rule, meta-

static foci do not develop. The symptoms are due more to absorption from the local necrotic focus than to the bacteriæmia.

The prognosis is almost always bad in these cases. Prophylactic treatment is most important, while extensive incisions and strenuous antiseptic applications are called for when the process is established.

During the course of any of the various types of septicæmia which have just been described, the symptoms of metastatic pus foci may develop and the symptoms of pyæmia will then be added to those of the existing septicæmia.

Pathological Anatomy.—In the most acute and severe forms of septicæmia the process is so rapid that few gross pathological lesions may be demonstrated. In such cases there are no special changes in the original wound. In the less severely acute cases, due to the action of pyogenic micro-parasites, the edges of the original wound first show inflammatory redness and become puffed up and swollen. If the wound is an open one the granulations look unhealthy and the wounded surfaces are covered with a fibrinous exudate. In these cases a foul, necrotic odor is usually absent. There are many exceptions to this rule, and cases are seen in which, within a few hours of the accident, the wound secretion is most foul. These cases are the most virulent we have to deal with, and generally terminate fatally within a few days. The common pus of a suppurating wound is odorless, and the presence of a necrotic odor generally signifies the presence of bacteria other than the staphylococcus and streptococcus. The local infection may be represented by a fairly large and extending area of putrefying or gangrenous tissue, or by an extensive septic phlegmon. The primary focus may be a carbuncle, an otitis media, an osteomyelitis, a pneumonia, or any suppurative process. The lymphatics are frequently involved, and varying degrees of lymphangitis and lymphadenitis are observed. In the more severe cases there is a rapid development of a severe anæmia. The bacteria and their toxins are present in the blood. The red blood cells are diminished in number and show degenerative changes. *Leucocytosis* is variable. In the severe and rapidly fatal cases there is little if any leucocytosis. In the chronic cases it is moderate, but in the subacute cases the leucocytosis is sometimes marked. The phenomena of thrombosis and embolism are not present.

After death decomposition sets in rapidly. The blood is dark, does not coagulate well, and quickly decomposes. The most constant changes are seen in the gastro-intestinal tract. Small ecchymotic spots may be seen, especially in the mucous membrane of the stomach, duodenum, and rectum. There is a marked gastro-intestinal inflammation, varying in degree with the severity of the toxinæmia. The solitary follicles and Peyer's patches are swollen, and they sometimes break down and form ulcers. If the intestinal changes are severe the serous covering of the intestines may share in the process and give rise to a cloudy or sero-sanguineous exudate, which collects in the peritoneal cavity. The pathological findings in the heart and lungs are variable. There may be small effusions

in the pericardial and pleural cavities. In such cases the effusion is apt to be cloudy. Small ecchymotic spots may be noted on the pericardium, endocardium, and pleura. Œdema of the lungs and hypostatic pneumonia frequently precede death. The spleen is almost constantly enlarged. Few changes are noted in the liver, aside from the so-called cloudy swelling. According to Hildebrand, there occur on the surface of the kidney small areas of hyperæmia, which he attributes to the heaping up of micro-parasites in the afferent vessels and within the capillaries of the glomeruli. There are present cloudy swelling of the kidney and a catarrhal inflammation of the urinary tract. The nervous system shows few changes.

PYÆMIA.

Pyæmia is a general infective disease of the body, characterized by a constitutional intoxication in which the signs and symptoms of metastases break in upon the general symptoms. It is not possible to differentiate etiologically between septicæmia and pyæmia. As a result of the most extensive experiments by many investigators, it has been discovered that the same micro-organisms may give rise to both conditions; and, further, that at one stage of the disease a patient may present all of the classical symptoms of septicæmia, and then suddenly, without any added etiological factor, the clinical signs and symptoms of a metastatic focus develop and a transition to pyæmia takes place.

The presence of the pus-producing microbes is essential to the development of pyæmia. The old theory, as advanced by Piorry, that the disease is always produced by the entrance of pus into the blood, has been exploded. It is true that when infected pus escapes into the general circulation pyæmia generally results, but in the majority of cases it is not due to such a cause. A primary focus of suppuration is the rule in pyæmia, but, just as is the case in septicæmia, this primary focus is not always demonstrable. From this primary focus the pyogenic microbes gain access to the circulation, and first bring about a general septicæmia. The microbes carried by the circulation may lodge in the parenchymatous organs and there bring about a secondary inflammatory process; suppuration then occurs, and a metastatic abscess develops. Or the condition may be brought about in a different way; that is, through the medium of a thrombus. If the primary suppurative process is in the immediate neighborhood of a large vein, the walls of the vein are apt to become involved. An inflammatory process develops in the perivascular spaces, and a round-celled infiltration of the adventitia and media occurs. The intima becomes swollen, a proliferation of endothelium occurs, and fibrin is deposited on it. This becomes the nucleus of a coagulum. Finally, the vein becomes occluded by an extension of this coagulum, and a thrombophlebitis is established. This process extends for a variable distance along the vein. From this thrombus small bits may be broken off, and, entering the general circulation as emboli, find lodgment in the various organs, plugging the smaller vessels and in this way producing infarcts. These in

turn may become infected by the bacteria in the circulating blood, thus giving rise to metastatic pus foci. Again, in the primary focus the microbes may invade the thrombus and bring about purulent softening of the mass. Portions of the thrombus break down and small particles, emboli, laden with bacteria, mix with the blood stream and pass through the heart into the lungs. The heart itself does not always escape, and a suppurative pericarditis or ulcerative endocarditis may be set up. The heart muscle itself is seldom invaded. In this way metastatic abscesses may be set up in almost every part of the body. In the lung a metastatic abscess generally is preceded by the formation of an infarct, due to the plugging of a terminal artery. If the artery—in some other part of the body, for example—is not a terminal artery, the infected embolus gives rise to an endarteritis and a localized abscess.

Many experiments have been tried to ascertain the probability of pus, when injected into the general circulation, giving rise to metastatic abscesses, and as a result of these experiments it has been discovered that only under certain conditions do metastatic abscesses develop. Only when large quantities of unfiltered necrotic stringy pus—*i.e.*, masses which acted virtually as emboli—were repeatedly injected did the metastatic foci develop. It can easily be understood that such conditions as these seldom occur in pyæmia in man. It seems much more probable that the bacteria circulating in the blood may become agglutinated into clumps, a number of these clumps coalescing and forming a plug, which stops up the small capillaries, and thus gives rise to a metastatic focus. This would explain many of the metastases which are found in the kidney, liver, muscles, etc.

There are certain conditions which predispose to the development of pyæmia. The disease seems to be more prevalent in overcrowded hospital wards, and in cities which contain many wounded soldiers, and, in general, in unsanitary localities. The anatomical structure of certain tissues predisposes to the disease, and suppurative inflammation of these tissues has long been looked upon as liable to develop into pyæmia. This is especially true of severe wounds of the bones of the skull and extremities, of wounds of the joints and of tendon sheaths, and of traumatic amputation wounds of the arms and legs; it is also true of wounds which involve the large veins.

Symptoms and Diagnosis.—As has been already stated, pyæmia may develop at any time in the course of a septicæmia, but in such cases the number of metastatic foci is generally limited and the appearance of the symptoms of new pus foci is simply incidental and does not in general alter the symptoms of the existing septicæmia. It is proposed here to describe a condition which is characterized by a somewhat different train of symptoms, presenting a distinctly different clinical picture.

As a rule, pyæmia develops during the period of suppuration in the wound. It may, however, develop before the local suppuration has taken place, owing to

a direct infection of the blood, or in the course of a chronic inflammation. The latter, however, is exceptional. There may be certain premonitory symptoms in the wound, such as its general appearance, a change in the character of its secretion, or the development of an extensive thrombo-phlebitis. The general system shows only a slight degree of intoxication at first, with some loss of appetite, general malaise, and a moderate fever. The disease itself first makes itself manifest by the occurrence of a severe chill. This is the rule, but there are

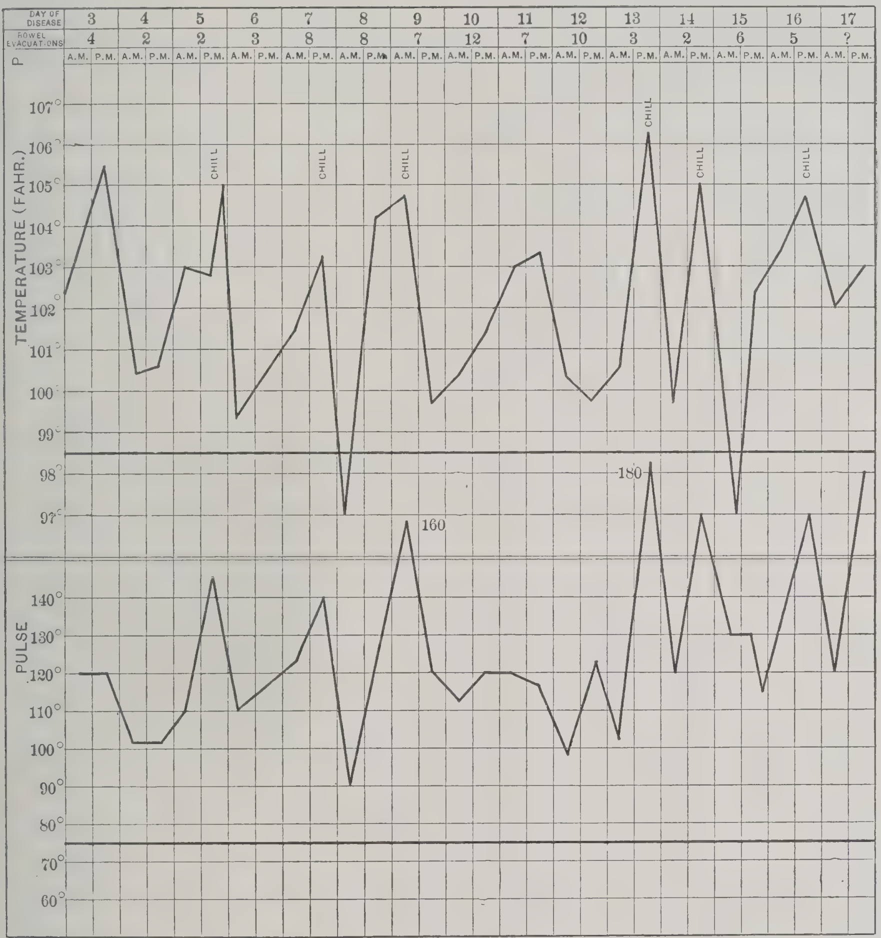


FIG. 132.—Typical Temperature Chart of a Case of Pyæmia. Primary focus in pelvis; metastatic foci in lung. Staphylococcus pyogenes demonstrated in the blood. The case was accompanied by marked gastro-intestinal symptoms.

many exceptions to it. The most characteristic symptom is the irregular course of the temperature, which rapidly changes from the highest point to the lowest point in a few hours. The relation of the chills to the course of the temperature is not always constant. Sometimes the chill is entirely absent, but even in these cases the temperature still shows the marked excursions which are so characteristic. As a rule, the initial chill is accompanied by a rapid rise in tempera-

ture, but in those cases in which a septic temperature already exists this rise is not so marked. The chill and rise in temperature may be repeated at irregular intervals during the day or night, or may recur regularly every day or every other day, thus simulating malaria. In the more acute cases the chill is repeated three or four times in a single day. Following the initial chill the temperature may gradually drop to normal and so remain for several days, to be again interrupted by the occurrence of a chill.

The course of the temperature is usually very irregular, and is intermittent or remittent in type. No two cases are alike, and in fact the daily temperature in the same case varies from hour to hour. Some cases will show for a few days a regular morning remission, but on the following day there will be a sharp morning rise to 104° F. or higher; or, during the course of the night there may be a chill and a sudden rise of temperature to 105° or over, followed in a few hours by a drop to a subnormal temperature. These wide excursions of temperature are especially characteristic of pyæmia. In exceptional cases there is a regularly remittent type of fever, which is only disturbed by the occurrence of chills. That a chill always means the establishment of a new metastatic focus has not been clearly proven, but it is held by the majority of writers to be the most probable explanation of the phenomenon.

The pulse at first is strong and full, and usually varies with the temperature, dropping even to normal. Later in the disease, it becomes rapid and weak, and does not show as great excursions as the temperature.

The general condition of the patient varies with the severity and the stage of the disease. The marked apathy and prostration seen in septicæmia are absent. The patient is painfully conscious of the severity of his disease, and shows anxiety as to his condition. Gradually he becomes weaker, and the effect of the toxins upon the brain may become more evident. Delirium may develop. Nausea and vomiting sometimes accompany the hyperpyrexia, and diarrhœa often occurs late in the disease. The tongue becomes dry and swollen, and the breath is foul. An especially characteristic feature of the disease is the yellowish discoloration of the skin, which may be due either to a destruction of the red blood corpuscles and a consequent deposit of pigment in the skin, a *hemorrhagic icterus,* or to inflammatory changes in the liver itself. The urine shows the usual changes due to an infective disease.

Of greatest importance, and a most characteristic feature of the disease, are the clinical symptoms of the development of *metastases* in the various organs and tissues of the body. In the acute and severe cases the lungs are the organs most frequently the seat of these metastatic abscesses. However, these foci may remain so small that they do not give rise to prominent symptoms, and, as is especially true in the chronic cases, the muscles, joints, and subcutaneous tissues often show metastatic foci before the lungs appear to be involved. It is probably true that in every case of pyæmia infarcts occur in one or more of the

internal organs. These may, however, remain microscopically small and not give rise to symptoms. In the severe forms the number of infarcts is generally large. In the chronic forms the number of secondary foci is generally small.

When the *lung* becomes the seat of a metastatic process, the first symptoms are an increase in the frequency of respiration and some dyspnœa. If the focus is situated in the substance of the lung it gives rise to the symptoms of a lobular pneumonia, which rapidly resolves itself into an abscess. If it is in the neighborhood of a bronchus, it may rupture into it and empty itself through the mouth, or it may rupture into the pleural cavity and give rise to the symptoms and sequelæ of a purulent pleuritis, empyema, or pyopneumothorax. More frequently, the metastasis takes place as an infarct immediately beneath the pleura, and a subpleural abscess results. The prognosis in these cases is bad.

The diagnosis of *liver metastases* is much more difficult. The occurrence of icterus does not necessarily point to an involvement of the liver. If the abscesses, which necessarily are small at first, are deeply seated, a diagnosis is often impossible. Only when the abscesses are situated near the anterior surface of the liver is it possible to diagnose them with certainty. Abscess and infarcts of the spleen and kidney are of less frequent occurrence, and a metastatic abscess of the brain is only exceptionally seen. The diagnosis of metastatic foci in the muscles, glands, and connective tissue is much easier. But even here the onset is often insidious, and the abscess may not be discovered until it is well advanced. Generally, however, the usual symptoms of a suppurative inflammation are apparent, and the diagnosis is established. Frequently the joints and bones become involved and give rise to characteristic symptoms.

In all of these secondary foci pyogenic microbes are found.

The course of the disease may be acute or chronic. In the *acute cases* the initial chill occurs early in the disease and is frequently repeated. The body temperature is very irregular, showing a characteristic curve, dropping at times from above 105° to below 96° F. The patient rapidly loses flesh and strength, and the skin reflects the profound changes in the blood. Grawitz reports a case in which after two days the red blood cells were reduced to 300,000 per cubic millimetre. The pulse becomes rapid and feeble, ranging from 110 to 160 per minute. The tongue is dry. The symptoms of metastases develop, but the abscesses do not reach any considerable size. The stools become frequent and are often blood-stained. Delirium develops and is followed by coma, and death takes place from heart failure, or suddenly from pulmonary embolism, often with a subnormal temperature.

The *chronic cases* may last for weeks, or even months. This may best be illustrated by an actual case which occurred in the Methodist Episcopal Hospital in Brooklyn.*

Patient, a few days previously, had suffered from an attack of follicular ton-

* Spence: Brooklyn Med. Jour., June, 1904.

sillitis. When first seen she complained of headache, general abdominal pain, and fever. Temperature, 103.6° F. Abdomen somewhat distended and tender. Leucocytes, 9,400. Widal test negative. *Second day*, slight epistaxis, vomiting, and chilly sensations. *Fourth day*, she had a distinct chill and complained of tenderness of the left forearm. Leucocytes, 9,000. Diazo reaction positive. Temperature irregular and ranging from 97.4° to 105.6°. Every day there were chills and profuse, clammy perspiration. General condition steadily growing worse. Slight tenderness on forearm continued, and on the *seventeenth day* indistinct fluctuation was elicited. An incision was made, and pus was found, dissecting its way between the flexor muscles. The pus showed a pure culture of Streptococcus pyogenes. On the *nineteenth day* a small collection of pus was discovered above the right scapula. This was evacuated. Both wounds did well. The chills and fever continued, however, and there was no change in the irregular up-and-down course of the temperature.

The patient became greatly emaciated, but her mental condition remained fair, and her appetite for the most of the time was excellent. A slight systolic murmur was heard at the base of the heart, but disappeared after a few days.

At the end of the *fourth week* the patient complained of pain in the right shoulder, and there was some tenderness on motion and when pressure was made over the joint. The temperature still continued its irregular course. During the *sixth week* she became restless and delirious at night. The pain in the shoulder continued with varying severity, and at the end of the *eighth week* the shoulder region became slightly swollen. An incision was made into the joint and pus was evacuated. A gradual improvement immediately followed. The temperature became lower, but did not remain normal until the *fifteenth week* of the disease.

At times there were points of tenderness over the upper and lower extremities and on the chest. There was no redness and very little swelling at those places where pus was found.

Albumin and casts were present in the urine, but disappeared with the other symptoms. The wound healed slowly and motion of the joint was gradually obtained.

This is a very fair example of a class of cases which we occasionally see—cases that run a chronic course with acute exacerbations and terminate in recovery.

The prognosis of pyæmia is almost always bad. In the acute cases all forms of treatment seem to be unavailing, and the patient dies in from one to three weeks. In the chronic cases the prognosis is somewhat better, but even here there are few instances of recovery, and then only after a long, protracted illness. When the metastatic abscesses involve the viscera the disease almost always proves fatal.

Septico-pyæmia.—Von Leube has described a special form of septic infection under this title, and although, from a bacteriological standpoint, it is caused by the same micro-organisms which give rise to septicæmia and pyæmia, still it should be clinically differentiated from them. The patient generally, without any known suppurative focus, gradually or suddenly develops a general septic infection, which is characterized by an irregular fever, a disproportionately high pulse frequence, great emaciation, marked nervous manifestations, and special symptoms referable to the various organs in which the septic poisons become localized. The fever often resembles that of a fluctuating typhoid, but may be continuously high. Chills are frequent. The heart is early affected, and in certain cases an inflammation of the endocardium is the first symptom of the disease. Von Leube believes that the so-called malignant endocarditis is only one of the manifestations of this disease. Inflammation of the joints and serous membranes is a prominent feature and of frequent occurrence. Symptoms referable to the nervous system, such as headache, vertigo, sleeplessness, delirium, convulsions, and temporary paralysis, are quite constant. Icterus is only occasionally seen. The kidney is markedly affected. Changes in the skin are almost constant. Roseola, erythema-like urticaria, purpuric spots, hemorrhagic pemphigus, blisters, pustules, herpes, etc., develop. Metastatic pus foci may occur anywhere. The disease runs an acute or subacute course, and almost without exception terminates fatally.

Pathological Anatomy.—The pathological changes which are found in individuals who have died from pyæmia are not always constant, but in general they are characteristic of the disease. The primary focus of suppuration may be entirely healed, but usually in the acute cases this is not the case, and we find evidences of a gangrenous or necrotic wound in the neighborhood of which the veins are inflamed and thrombosed. In the chronic cases, especially those of cryptogenetic origin, there are evidences of multiple abscesses. The heart seldom escapes entirely, and frequently a purulent pericarditis, an ulcerative endocarditis, or chronic changes in the valves, are found.

The most constant changes are found in the lungs. They are, for the most part, due to the lodgment of emboli in the cortex of the lung, most frequently in the lower lobes. These emboli plug the terminal arteries and produce wedge-shaped infarcts. The embolus either contains pyogenic micro-organisms or becomes secondarily infected and a thrombus is formed. A septic endarteritis is set up, which in turn infects the infarct, and a circumscribed abscess results, which lies directly beneath the pleura and may give rise to a pleuritis, empyema, pyopneumothorax, etc. However, such is not generally the case, the abscess remaining localized. Other inflammatory foci may also be established in the lung independently of emboli, and lobular pneumonia then results.

The liver is frequently the seat of metastatic abscesses. They vary in size from microscopic accumulations of pus cells to an abscess which may destroy an

entire lobe. Usually they are not due to emboli, but to an accumulation of micro-parasites in the smallest capillaries. The infection may spread to the portal vein and cause there a thrombophlebitis.

The kidney is occasionally the seat of metastatic abscesses, but more frequently there will be found a catarrhal inflammation and cloudy swelling. The spleen is usually enlarged and soft. Brain abscesses are not frequent, but may occur together with a purulent meningitis. In the joints may be found all forms of suppuration, but generally not of a severe type. The knee, ankle, and shoulder joints are the ones generally affected. The skin also may be the seat of some inflammatory disturbance, the severity of which varies from a mere erythema, which disappears in a few days, to a subcutaneous abscess. All or any of the other tissues and organs may be invaded.

In the *chronic forms* of the disease various degenerative changes are seen. There may be amyloid degeneration of the liver, spleen, and kidney, fatty degeneration of the heart, and chronic changes in the valves. The lung shows the scars of many infarcts and abscesses. There is sometimes a chronic exudative pleuritis. A chronic enteritis is also apt to be present, and various atrophic changes are seen in the kidney. The patient is greatly reduced in weight.

TREATMENT OF SEPTICÆMIA AND PYÆMIA.

The treatment of all the various forms of general septic infection resolves itself into an attack upon the pathogenic bacteria which give rise to the disease, in order to destroy them and limit their activity, and to remove from the system their toxins and the results of their action. There is no other surgical condition in which a careful prophylaxis plays so important a part as it does in this disease.

Aseptic and antiseptic surgery are two distinctly different things. The former has to do with the prevention of infection by excluding all bacteria from the wound. The latter deals more with the prevention of the growth of bacteria in a wound by the use of certain chemicals which kill or attenuate the micro-organisms. The primary prophylaxis of septicæmia and pyæmia is included under the consideration of these subjects, and has to do with the preparation of the patient and the surgeon for the operation, the sterilization of the instruments, dressings, etc., and the treatment of wounds in general. The general hygiene of the patient and the sick-room, the prevention of infection in various surgical diseases and conditions, and the treatment of infected wounds will be discussed by other writers, and it is therefore not necessary that I should consider these subjects in detail in the present article.

Many surgeons seem to overlook the importance of preventing secondary infection or a mixed infection in a wound. It is just as important, for the prevention of a general septicæmia, to guard against a secondary infection in a wound

as it is to prevent primary infection in a clean wound. If the presence of infection in a wound is discovered early enough and prompt measures are taken properly to drain the wound and all the foci of suppuration, the chances are that general sepsis will never occur. In the presence of a surgical injury or any condition in which the vitality of a part is lowered, the most stringent aseptic measures must be adopted to prevent infection. Infections of necrotic or gangrenous areas are especially apt to spread rapidly and cause a general septicæmia. In the treatment of compound fractures, extensive lacerated and contused wounds, and burns, asepsis should be carefully carried out.

In the treatment of sapræmia the removal of the putrefying material and general stimulation are, in most cases, followed by recovery.

When septicæmia has developed, an immediate inspection of the wound must be made; indeed, it should be thoroughly explored. All pus foci must be evacuated and liberal incisions made, in order to establish a free drainage of the wound. The wound should not be packed with antiseptic gauze, as is so often advocated, for this retards the evacuation of the wound secretion. Large drainage tubes should be used and the wound frequently irrigated. Wherever possible, continuous irrigation is called for. When pyæmia has developed, the secondary foci must be sought for, and when abscesses form they should be opened, if possible, and treated in the same manner as the primary focus. Fochier observed that when active suppuration occurred in pyæmia and septicæmia the general condition usually improved. He therefore tried experimentally the *production of artificial suppuration* in such cases. This may be done by injecting subcutaneously from 2 c.c. to 5 c.c. of rectified turpentine. There is thus created an abscess in which the pus is sterile. In six of the cases in which he tried this experiment the results were favorable: a general improvement occurred, and the temperature was lowered. Trials made by other surgeons have not, however, met with much success.

There are a number of surgical conditions which specially call for early interference. Suppurative osteomyelitis should be treated early and radically. Certain rapidly spreading infections of the extremities call for amputation as the only means of saving life.

Klebs first suggested the ligature and removal of veins which contained infected thrombi, before the emboli should be broken off and set up metastatic foci. This procedure is especially applicable to thrombophlebitis of the sigmoid and lateral sinuses and jugular vein, arising from suppuration in the middle ear. The first step in the operation is the ligation and excision of a portion of the jugular vein. Then the sinuses may be exposed and the infected material thoroughly removed. This procedure, as a prophylactic measure, has met with success in the hands of some surgeons, but it will limit the disease only when it is due to the lodgment of infected emboli. The same principle has been suggested and tried in cases of thrombophlebitis of the portal vein.

In the cases of septicæmia arising from general peritonitis, the first indication is to clean out, as far as possible, the pus and serum accumulated in the peritoneal cavity. The second indication, of no less importance, is to clean out the intestinal tract and establish active peristalsis. The method originated by the writer in such cases is as follows: The first portion of small intestine which presents itself in the wound is taken and a purse-string suture is introduced. This suture should pass through the serous and muscular layers and should include an area the size of a ten-cent piece. An incision is made within this area and a small-sized glass catheter is introduced through it into the lumen, being retained in place by tying the suture. Gas and fecal material are allowed to escape. Then, through the catheter, the intestine is thoroughly irrigated, as far as is possible, with hot saline solution. After this has been done, three ounces of magnesium sulphate is introduced through the catheter into the lower part of the intestine, and the end of the catheter closed. During the first twenty-four hours the intestine is irrigated every three or four hours. Magnesium sulphate is injected daily and the irrigations are continued as long and as frequently as indicated. In this way it is possible to establish active peristalsis and aid very materially in the elimination of the toxic materials from the system.

The local treatment in *puerperal septicæmia* is a broad question and can be only briefly alluded to here. Where the infected process is localized in the uterus, the usual methods of treatment are curettage, hot intra-uterine irrigations, vaginal douches with antiseptic solutions, and the application of an ice bag. These measures, combined with the constitutional treatment to be described below, are generally followed by a cure. When the process is not confined within the uterus, more drastic measures have been advocated. Tuffier* says "that in a given case of septicæmia, post-partum or post-abortum, when no cause for the fever can be found either in the external genitals or in other organs, when the usual methods of treatment are of no avail, when the peritoneum and adnexa are intact, and the uterus is large, flabby, and is discharging fetid lochia, and if the patient's general condition warrants it, total extirpation of the uterus should be done, whether there be placental retention, a sloughing myoma, or the so-called metritis dissecans." Many surgeons, however, do not agree with this. The late Dr. Pryor advocated splitting the posterior lip of the cervix, thus providing for a more thorough drainage of the uterus, opening the cul-de-sac of Douglass, and packing it with iodoform gauze. He held that the iodine set free and absorbed by the tissues acted as a powerful local and general antiseptic.

The same principle applies in all cases. Wherever possible, limit the local activity of the micro-organisms.

General Treatment.—The first indication in the general treatment of the patient is to foster and stimulate in every way the excretory organs of the body.

* American Gynæcology, January, 1903.

The bowels should be made to act regularly, and the rectum should be emptied by daily enemata, if necessary. Diaphoresis and diuresis should be stimulated. The general hygiene and nursing of the patient are of the utmost importance. The patient's body must be frequently bathed, and he should be given every opportunity to obtain fresh air and sunlight. The nourishment of the patient is important. Small quantities of food given at frequent intervals will be best borne by the stomach. If the patient refuses food or cannot retain it, rectal enemata must be given. The rectum should be gently washed out, and then an enema containing peptonized milk, peptonized eggs, and whiskey (not exceeding four or five ounces in amount), should be given every four hours.

Drugs.—In general, drugs given to affect the course of the disease are not of much avail. Some physicians believe that calx sulphurata, given in ten-grain doses every three hours, will retard pus formation. Alcohol is the one drug which seems to have a beneficial effect in septic conditions. The patients stand it well, and it should be given in small doses frequently repeated. Symptomatically, a number of drugs are called for. Heart stimulants, such as tincture of digitalis, caffeine, and strophanthus, are often indicated, as well as drugs to decrease the gastro-intestinal inflammation. Nerve sedatives may be necessary.

Decinormal Salt Solution.—The use of this solution in various ways is unquestionably of benefit to the patient. Wernitz recommends its use in the form of hot rectal irrigations, to be given through a high rectal tube. The solution is allowed to flow in gently, and the procedure is continued until the patient shows discomfort, or until the return flow is clear, showing that the lower gut is entirely clean. This is repeated frequently during the twenty-four hours. Wernitz claims that it is followed by a falling of the temperature, profuse diaphoresis, increased diuresis, less thirst, and a general improvement in the patient's condition. Repeated submammary or subcutaneous injections of decinormal salt solution may be given. It is rapidly absorbed and acts as an effective cardiac stimulant, increasing diaphoresis and diuresis, and often having a distinct sedative effect upon the patient. Intravenous infusions may also be indicated, and may be frequently repeated. Such infusions are almost always followed by beneficial results, and it is probable that they aid materially in the elimination of the toxic material from the tissues and the blood.

Serum Therapy.—There are two distinct types of sera used in septicæmia. The one acts by virtue of its antitoxic properties, and the other acts as a bactericide. Antidiphtheritic serum is an antitoxin, and has no effect upon the micro-organisms. It has been shown that the diphtheria germs can be made to grow on diphtheria antitoxic serum. The antistreptococcic serum, on the other hand, acts as a bactericide and has little antitoxic action.

It has been definitely established that diphtheria antitoxin is of distinct value in the treatment of septicæmia caused by the micro-organisms of diphtheria. It is a specific. This is made possible by the fact that the same organism is always

the cause of the disease. When we study the streptococci, on the other hand, we find that all streptococci are not the same; that is, there seem to be numerous varieties of streptococci which cannot be differentiated from one another. These different varieties vary also in respect to their virulence, so that when a streptococcic serum is produced it will be found to vary in proportion to the virulence and variety of the streptococcus used in the immunization.* It is not strange, then, that the results of the use of this serum should vary greatly.

The use of antistreptococcus serum is indicated in those cases in which we have a pure infection of the streptococcus. When there is a mixed infection, it acts only upon the streptococcic factor of the disease. In 1902 Packard and Wilson† collected 117 cases in which the serum had been used, and they report recovery or marked improvement in 114 of the cases.

Bumm, quoted by Young,‡ concludes that the employment of antistreptococcus serum, when a general peritonitis of puerperal origin, a pyæmia, a parametritic phlegmon, etc., exist, is ineffectual and useless. He believes it is of use in the early stages where the organism has not extended beyond the endometrium, or where an extensive bacteriæmia does not exist.

The serum should be injected early. The dose varies from 10 c.c., repeated twice daily, to 25 c.c. or more, injected every second or third day.

Intravascular Antisepsis.—Since the disease is essentially a blood disease, surgeons have long sought for some antiseptic solution which could be injected into the circulation and destroy the organisms without doing injury to the patient. Credé was one of the first to experiment along this line. He did not at first inject substances directly into the circulation, but caused a local hyperæmia of the skin, and then, using a fifteen-per-cent ointment of colloidal silver, rubbed 2 or 3 gm. of the same into the hyperæmic area. This is absorbed by the blood and attacks the micro-organisms. Later, he injected from 2 to 10 c.c. of a two-per-cent solution of collargol intravenously. He claims that it is a non-irritating, strongly bactericidal agent, and that by its employment marked improvement and often recovery follow in even the most severe forms of septic poisoning. Many surgeons have tried this method, and are divided in their opinions as to its efficacy.

Maguire, of London, experimented by injecting a solution of formaldehyde gas directly into the circulation. His conclusions were that 50 c.c. of a 1 in 2,000 solution of formaldehyde—that is, a 1 in 800 solution of formalin—was the maximum dose that could be safely injected in man. In 1903 a number of cases of advanced septicæmia were treated by injecting this solution intravenously. The results at first were encouraging, but, owing to the dangers and uncertainty of the method, it has not come into general use.

* Travel: Klinisch-therap. Wochenschrift, 1902.
† Amer. Jour. of the Med. Sciences, December, 1902.
‡ Boston Med. and Surg. Journal, Aug., 1905, p. 216.

In chronic cases most attention must be given to the nourishment and general stimulation of the patient. Good, nourishing food, fresh air, and hygienic surroundings are essential.

ERYSIPELAS.

Erysipelas is an acute, inflammatory disease of the skin or mucous membrane, caused by infection of a wound of the skin or mucous membrane by the streptococcus of Fehleisen. It is characterized by a peculiar, non-suppurative inflammation or dermatitis, which begins in the wound and rapidly spreads over the skin or mucous membrane, and which usually is self-limiting and ends in resolution. It is accompanied by fever.

Etiology.—It is generally accepted that erysipelas always arises from an infection of a wound with streptococci. The so-called idiopathic cases, most frequently seen on the face, are not due to infection from within, but in every case there must be some break in the continuity of the epithelium covering the skin or mucous membrane. The original wound may be simply an abrasion or slight pin prick, and may be entirely healed before the symptoms of erysipelas develop; nevertheless, a wound of some sort must have existed. In surgical practice erysipelas is most frequently seen as a complication of infected wounds and in debilitated, alcoholic, or other patients, in whom the resisting powers of the individual are diminished.

The existence of a specific micro-organism is still in question. The opinion of many bacteriologists is that it is always caused by the streptococcus of Fehleisen. However, there is a diversity of opinion on this subject. Welch* states that "the streptococcus of erysipelas does not differ in morphological or cultural characters from the Streptococcus pyogenes. The same pathogenic effects may be produced by each in animals and man, so that the weight of evidence is in favor of the identity of the Streptococcus erysipelatis with the Streptococcus pyogenes." Clinically, the two manifest themselves in distinctly different ways, and give rise to different pathological processes.

In erysipelatous inflammation the streptococci are found chiefly in the lymph capillaries and lymph spaces of the skin and subcutaneous fat. Here they multiply rapidly, and often are seen completely filling the lumen of lymphatic vessels. They are generally found most abundantly in the peripheral margins of the inflammation. If the inflammation penetrates more deeply, which is unusual, the micro-organisms are found in the connective-tissue spaces.

The occurrence of suppuration in the course of erysipelas is considered by many to be due to a mixed infection. If we accept the theory that the Streptococcus erysipelatis and the Streptococcus pyogenes are identical, then the suppuration is caused by mono-infection. If we believe that the Streptococcus erysipelatis is a non-pyogenic micro-organism, then the occurrence of a phlegmonous inflammation must be due to a mixed infection.

*Dennis: "System of Surgery."

The micro-organisms seldom gain entrance to the general circulation, and the constitutional symptoms are due to resorption of the toxins from the seat of infection.

Erysipelas is a highly infectious disease, and may be carried by instruments, the hands, clothing, dressings, etc., from one patient to another. One attack does not protect against a second attack. In fact, some people seem to have an especial predisposition to contract the disease, but subsequent attacks are not usually so severe as the primary one.

Symptoms.—The period of incubation varies. Generally it is short, from fifteen to sixty hours, or it may last for from three to seven days (Butler). During this stage there may be indefinite prodromal symptoms, such as headache, anorexia, or general malaise. Usually the symptoms are ushered in by a severe chill, which may be repeated. This is followed by a rapid rise of temperature (104° or 105° F.), accompanied by anorexia, vomiting, and, in debilitated patients, marked depression. At the same time, or within a few hours, the vicinity of the wound is seen to be swollen and red. At first, there is nothing characteristic about this redness and swelling. Frequently, at first, small red streaks may be seen to radiate from the wound, corresponding to the position of the lymphatics. This is best seen when the disease attacks the extremities. Later, these stripes disappear. The redness rapidly extends and involves a considerable area. The lymph nodes are more or less swollen. There are subjective symptoms of itching, burning, heat, and pain in the affected area.

Usually the *erysipelatous patch* is sharply circumscribed. It is elevated, irregular in contour, rose-colored or of a bright reddish color, and presents a smooth, glazed appearance. When pressure is made with the finger on the hyperæmic area, the redness disappears for a moment and the skin shows a peculiar, yellowish discoloration. The swollen tissues do not pit on pressure. The affected part is tender and feels somewhat like leather. In anæmic, cachectic individuals the redness is not so marked. The amount of swelling varies with the severity of the infection and the part affected. When there is much loose connective tissue the swelling is more marked. Often on the surface of the skin vesicles appear in large numbers. Several of these may run together and form large bullæ. These vesicles and bullæ contain clear or slightly cloudy serum, and may contain pus. As the inflammation advances they dry up and form crusts, or they may open and leave an ulcerated surface.

The inflammation is progressive and shows a great tendency to spread. On the face it usually develops on or near the nose, and spreads laterally along the lower border of the orbit. In men it has often been noticed that it stops where the beard begins on the cheek. It may pass up over the forehead and invade the scalp. In the more severe forms the eyelids become greatly swollen and close up the eyes. The disease is most active at the periphery of the inflamed patch, and, while it is thus spreading at the periphery, it may be subsiding in the central

portion. It may further extend and involve the neck. In facial erysipelas there is always danger of the infection penetrating deeply and causing meningitis. It may spread from the skin and involve the mucous membrane of the nose and mouth, and cases of pulmonary erysipelas have been reported. In severe cases the face and head may become enormously swollen and distorted, and such cases are accompanied by grave constitutional symptoms.

The inflammation may not be confined to any one portion of the body. It may start on the arm or leg and, steadily and irregularly advancing at the periphery, creep up over the shoulder or thigh and spread over the body. At the same time at the original site the inflammation may subside and entirely disappear, or, after temporary subsidence, it may start up again. Such a condition, called *erysipelas migrans* or *ambulans,* may cover a period of weeks. The constitutional symptoms are not generally marked.

Only exceptionally the erysipelatous inflammation is associated with suppuration. The pyogenic process may be superficial and result in the formation of pustules, or the infection may involve the deeper structures and set up a phlegmonous inflammation. This does not differ in its essentials from the phlegmonous inflammations already described. The infection may involve any of the deeper structures, and there have been reported cases, not only of periostitis and osteomyelitis, but also of joint involvement. Occasionally, small areas of necrosis and small localized abscesses may form throughout the erysipelatous patch. All grades of inflammation are seen, from the mildest dermatitis to the most severe form of acute purulent œdema or gangrène foudroyante of Maisonneuve.

The *constitutional symptoms* vary, as a rule, in proportion to the intensity of the local disturbance. This, however, is not constant. Sometimes large areas of skin are involved without marked systemic symptoms. The body temperature, as a rule, is characteristic. Following the initial chill there is a sharp rise. The first day the temperature may be only 103° F. The next morning there is no remission, but the temperature is higher, reaching 104°. This absence of morning remissions is considered by some to be pathognomonic. The temperature continues high for six or seven days, and generally terminates by crisis. Often, however, the drop to normal is more gradual. In exceptional cases, for the first few days the temperature may not be high, even though the typical local symptoms be well developed. At times the temperature is very irregular. Following the initial chill it may reach 106° or more, to be followed the next morning by a drop of three or four degrees. Again, it may follow the type of a severe remittent or intermittent fever, or an irregularly remittent fever. Such irregularities of temperature are not necessarily associated with suppuration. The temperature usually subsides before the local symptoms disappear.

The disease is almost without exception ushered in by a chill. It is generally severe and lasts for some time. Or the chill may be less severe and be repeated.

The chills may be repeated on the following day, or every day. They do not necessarily indicate the involvement of fresh tissue.

The *pulse* is increased in frequency and usually varies in proportion to the height of the temperature, seldom becoming very rapid except in the severe cases. The gastric symptoms are often more prominent at the beginning of an attack. The patient's tongue is thickly coated. Loss of appetite, nausea, and repeated

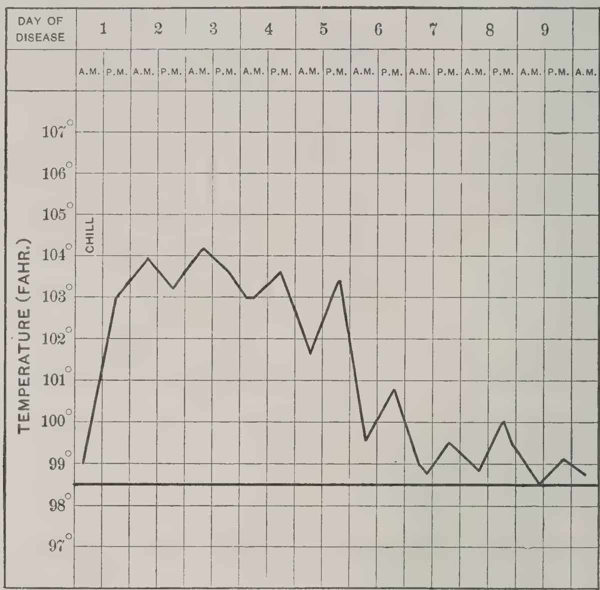


Fig. 133.—Typical Temperature Curve of a Mild Case of Facial Erysipelas.

vomiting are the rule. Diarrhœa is not a constant symptom. Often severe symptoms referable to the nervous system are observed. Headache, restlessness, delirium, and coma occur. These are more pronounced in the cases of facial erysipelas.

In the most severe cases all the constitutional symptoms of a general intoxication appear. High fever, dry, crusted tongue, alternating delirium and coma, involuntary evacuation of the bladder and rectum precede a fatal termination.

In erysipelas of the phlegmonous type, chills often recur and the temperature assumes a more typhoid type.

When the disease attacks the mucous membrane lining the air passages, marked swelling of the tissues takes place and fatal œdema of the glottis may occur.

Numerous complications may arise during the course of the disease. Among the most common may be mentioned meningitis, peritonitis, pleuritis, otitis media, nephritis, and pneumonia; also septicæmia and pyæmia.

Diagnosis.—In a well-developed case of erysipelas the diagnosis is often easy. The initial chill, with the rapid development of a continuously high temperature, together with the marked swelling and characteristic rose color of the sharply defined erysipelatous inflammation, are pathognomonic. Where the symptoms, however, are not so characteristic, the diagnosis is often difficult. Sometimes,

when the disease develops in the presence of other infections, a diagnosis is impossible. Occasionally we see cases of *chronic dermatitis*, with redness and thickening of the skin, which simulate erysipelas. In such cases there is no tendency, on the part of the disease, to spread; and, besides, the course is not acute, the area affected is less sharply defined, and there is no fever. There are certain forms of *acute dermatitis* which are not so easily distinguished from erysipelas. Especially is this true of the dermatitis due to the irritation of antiseptic solutions, such as dilute carbolic acid, sublimate solution, etc. The dermatitis in such cases is often most severe in the vicinity of the wound, and at first may simulate an erysipelatous inflammation, especially in the presence of a septic temperature. The inflammation does not spread so rapidly and is not so sharply circumscribed. The characteristic swelling and glazed appearance are absent. Erysipelas may be confused with a *progressive phlegmonous inflammation*, but in the latter the inflammation does not usually affect the skin and does not present the sharply defined, raised edges seen in erysipelas. In simple *lymphangitis* the inflammation generally follows the course of the larger lymph vessels, there is no typical inflammation of the skin, and the diagnosis is not difficult.

In *traumatic erysipelas*, if the wound be large, the erysipelatous inflammation usually is seen to start from one portion of the wound and send out fan-like projections into the normal skin. It has been likened to the flame from a gas jet, which it resembles in contour. The dermatitis does not, as a rule, involve the entire wound symmetrically, although this may take place. There is not much change in the appearance of the wound itself. The secretion may be diminished and a general improvement may be noted. Another point of diagnostic interest is the fact that the dermatitis does not necessarily spread in the direction of the flow of lymph in the lymph vessels. The lymphangitis may advance against the stream, and the vessels often become entirely plugged with micro-organisms, which multiply rapidly by division.

There is no constant relation between the subjective symptoms and the height of the body temperature. Patients are often seen with a well-developed erysipelatous dermatitis and with a temperature of 104° or 105° F., without any marked constitutional disturbance. Such a condition, however, is seen only in otherwise strong and healthy persons.

Erythema may simulate an erysipelatous dermatitis, but the inflamed area is not continuous, and areas of healthy tissue appear between the erythematous patches. The temperature, course, and constitutional symptoms help to differentiate the two.

In former years erysipelas was frequently seen as a complication of hospital gangrene, especially the diphtheritic form, also as a complication of diphtheritic inflammations of the throat and other mucous membranes. In these cases the inflammation of the mucous membrane was usually of a gangrenous type, and it is questionable whether the original infection was due to the streptococcus of erysipelas or whether it occurred as a secondary infection. Such involvements of the

mucous membranes are always attended with great swelling and diffuse redness, which are not especially characteristic and make the diagnosis most difficult.

The relation of erysipelas to pyæmia is still a much disputed question. Pyæmia does sometimes occur during the course of the disease, secondary foci becoming established in the lungs and other tissues, and giving rise to inflammation and suppuration. It is most frequently observed in the phlegmonous and gangrenous types of erysipelas, also in the cases of erysipelas which develop in large, freshly made wounds which easily develop thrombophlebitis. A thrombus may become secondarily infected by the erysipelas micro-organisms and so occasion pyæmia. The same is true of the relation between erysipelas and septicæmia. A fatal septicæmia, which does not differ from the forms of septicæmia already described, may at any time develop.

Pathological Anatomy.—In the milder cases the erysipelatous inflammation is most frequently confined to the skin, but may invade the underlying connective tissue. The blood-vessels are markedly dilated and are crowded with cells. There is a varying degree of serous exudation. The superficial layers of the epidermis are raised in places, forming vesicles. The cells of the rete Malpighi are at first swollen and enlarged, and become vacuolated, but later they shrink and are partly destroyed. The serous exudate invades the hair follicles, and as a result the hair becomes separated from its papilla and drops out. There occurs a rapid and profuse, small-celled infiltration, which is first observed in the cutis and subcutaneous cellular tissue (Volkmann). This round-celled infiltration is especially marked around the lymph vessels, and may remain after the bacteria disappear. The lymph vessels become filled with streptococci, which often entirely plug the vessels. According to Tillmanns,* the small-celled infiltration becomes crowded together in the cutis and subcutaneous tissues, and often forms small, microscopical abscesses. He believes that this takes place more frequently than is generally recognized; further, that the most active stage is found in the outlying regions of the erysipelatous patch, and sometimes, more especially in the cases complicated with pyæmia, during the acme of the disease the connective tissue, the lymph-, and small blood-vessels are filled with streptococcic vegetations. The cocci often extend as a fine network through the tissues.

It is not always possible, however, to demonstrate the presence of the bacteria. They have frequently been found in the circulating blood.

In the typical cases the local symptoms subside after three or four days. The round-celled infiltration disappears rapidly, while the serous exudate is removed more slowly. In the phlegmonous type of the disease the changes are naturally more extensive and are accompanied by suppuration and loss of tissue.

The secondary systemic changes are not characteristic, and resemble those which usually accompany all acute febrile infectious diseases. Degenerative alterations are seen in the blood and the vessels.

* "Deutsche Chirurgie," Lieferung 5.

Desquamation usually takes place.

Prognosis.—As a rule, the prognosis is favorable, but it varies with the severity of the infection and the general condition of the patient who is attacked. In general, the mild cases run their course in about a week and the disease then subsides. The greatest danger arises from the complications, such as meningitis, œdema of the glottis, pyæmia, etc., which have already been mentioned.

Treatment.—When we consider the etiology and pathology of erysipelas, we find that it is a disease due to the activity of streptococci which do not differ morphologically and physiologically from the ordinary pus-producing streptococci, and that under ordinary conditions they give rise to an inflammatory process which is usually localized and tends to be self-limiting; further, that when the disease invades the general system the symptoms resemble those produced by other streptococci. Finally, we believe that the constitutional symptoms are due to the resorption of the toxins and other poisonous products elaborated by the bacteria, and to the effects produced by the action of the bacterial toxins on the tissues. In other words, the conditions are similar to those which are found in other wound infections, and therefore, aside from the local indications and serum therapy, the general treatment should be the same as is employed in the other forms of wound infections.

Prophylaxis is of greatest importance. The disease is highly infectious, and is communicable by means of any object or medium which will convey the bacteria to a wound. On this account the patient must be isolated, and nothing which touches or is in the vicinity of the patient should escape disinfection.

General Treatment.—Good nursing, pure air, and careful attention to hygienic surroundings are important. The bowels should be regulated and the diet should consist chiefly of milk, broths, and eggs. Stimulants are sometimes called for. Alcohol may be given in the form of whiskey or brandy. Alcoholic beverages, such as beer and champagne, may be used in suitable cases. The chloride of iron, in doses of twenty to forty drops every two hours, has been extensively used, but not so much now as in former years. As a general rule, the use of antipyretics is contraindicated. Salinger strongly recommends the hypodermic injection of pilocarpine. The drug is administered until the physiological effects are produced. All drug treatment, however, is unsatisfactory.

Local Treatment.—The various methods of local treatment are all based upon the general principles of antisepsis, with the idea of allaying the local inflammation and checking the spread of the disease. Of all the antiseptic solutions used, those of corrosive sublimate, 1 in 1,000, and carbolic acid in varying strengths, have met with the greatest favor. Corrosive sublimate may be employed in the form of compresses kept moist with the solution and applied to the erysipelatous area, or it may be injected hypodermatically around the edge of the area. Carbolic-acid solutions may be similarly used. Kraske advocated scarification of the skin at the periphery of the inflamed area, and then the application of a warm

solution of corrosive sublimate. Some writers recommend painting the diseased area and the surrounding skin with tincture of iodine. Others apply compresses kept moist with alcohol, sodium hyposulphite, potassium-permanganate solution, etc. Cebrian advocates painting the affected area twice daily with a ten-per-cent solution of ichthyol in collodion. In the experience of the writer the use of a ten- to twenty-per-cent ointment of ichthyol, mixed with an equal quantity of vaseline or lanolin, has been found of advantage. The diseased area is first carefully cleaned with soap and warm water, and then the ointment is applied.

When suppuration occurs, incision and evacuation of the pus are called for, and the wound should be treated as set forth in the chapter on that subject.

Serum Therapy.—Antistreptococcus serum has been employed for over a decade in the treatment of erysipelas, but such are the vicissitudes of the disease that it is impossible to say accurately how much good comes of its use.

In 1895 Marmorek reported a series of 306 cases of erysipelas, in 165 of which antistreptococcus serum was used. Prior to this, the average mortality was assumed to be about 5.12 per cent. The dose varied from 10 to 20 c.c. In this series of cases the mortality was 1.63 per cent. When a weaker serum was used,

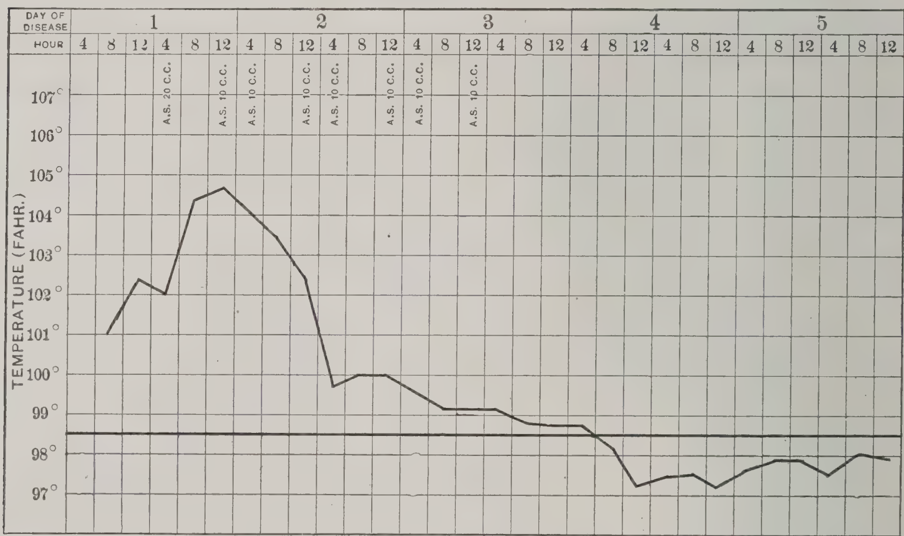


FIG. 134.—Temperature Curve of a Case of Facial Erysipelas, Showing the Effect of Injections of Antistreptococcus Serum.

the per cent rose to 4.82. After the injections the symptoms diminished markedly within a few hours, and a general improvement was noted.

Ayer* reports a series of fifteen cases treated by injections of antistreptococcus serum. He concludes, from a careful study of these cases, as compared with a series of seventy-nine typical cases in which the serum was not used, that, if the treatment is begun early enough, the course of the disease is considerably shortened, its extension is inhibited, and that there is a striking beneficial effect

*Medical Record, March 4th, 1905.

upon the general condition of the patient, the temperature, pain, and discomfort incidental to the disease being reduced; further, that its use is attended with no danger, even in large doses, and that it rapidly reduces the pathological leucocytosis and prevents or suppresses febrile albuminuria.

The efficacy of the treatment depends upon the promptness with which it is applied. As a rule, it is best to use small doses frequently repeated. If the case is seen on the first or second day of the disease, 10 c.c. should be injected and repeated every four hours for four or five doses. If the patient is seen first on the third or fourth day of the disease, larger doses should be given.

From all sides good results have been reported from the use of the remedy.

The curative influence of erysipelas on various diseases has been the subject of much investigation. Certain chronic diseases, especially of the skin and joints, have been favorably affected by an attack of erysipelas. Cases of carcinoma and sarcoma which have been cured in this way have been reported.

TETANUS.

Tetanus is an infectious disease due to a specific organism, the tetanus bacillus, and is characterized by intense spasms and painful contractions of certain definite muscle groups. It may be either acute or chronic in its course.

Two varieties have been described—*traumatic tetanus*, due to infection of a wound with the tetanus bacillus; and *cryptogenetic tetanus*, in which the characteristic symptoms of tetanus develop without a discoverable local focus of infection. The term idiopathic, when used in such a connection, is especially objectionable, because it indicates that the disease may be due to an unknown cause, whereas we know definitely that tetanus is always caused by a certain specific organism, and never occurs unless the specific micro-organism gains access to the tissues.

Etiology.—As has been already stated, the disease is due to infection with the tetanus bacillus. The micro-organism is a strict anaërobe, and cannot be made to grow in the presence of oxygen. It was first obtained in pure culture by Kitasato in 1889, but was previously described by Nicolaier in 1884. It is found widely distributed in the soil, especially in barnyards and in soil which has been repeatedly fertilized with manure. Certain localities seem to be especially favorable for its growth, as has been demonstrated in certain districts of Long Island, N. Y. It has been frequently found in the intestinal discharges of the horse, and sometimes of man. It would seem, from numerous experiments and the observation of actual cases in man, that the disease does not always develop when the micro-organism gains entrance to a wound. It probably follows the same laws of development that have been observed in infectious diseases due to other micro-organisms. Wounds which are accompanied by extensive laceration and destruction of tissue present favorable media for the development and growth of the germs. Suppuration due to a mixed infection favors rather than hinders the activity of the bacilli. Many cases of tetanus developing after supposedly aseptic

surgical operations have been reported. In such cases the carrier of the infection has often been thought to be the catgut or other suture material used.

It has been observed that certain climatic influences, mixed infection, anaërobic conditions, and burns favor the development of the disease. Gunshot wounds and the usual Fourth-of-July casualties generally present the last two conditions, and often produce a mixed infection as well. The bullet, powder, wadding, etc., are projected into the body and penetrate deeply, often becoming so buried in the tissues that air is excluded. Local destruction of tissue takes place and hæmatomata are formed. There is greater danger from blank cartridges than there is from ball cartridges. Dr. Connolly, of Newark, recently examined blank cartridges, such as are used to celebrate the Fourth of July, and found that they contained the germs of tetanus. Dr. Park, of the New York Board of Health, made extensive investigations along the same line, and was unable to find the bacilli in the blank cartridges, and he concluded that the infection, under these circumstances, was due to the fact that fragments of skin more or less covered with dirt were driven into the wound. Fourth-of-July accidents are the most prolific cause of tetanus in the United States. More than half the deaths due to tetanus are caused in this way. In a statistical report of 415 deaths from Fourth-of-July tetanus, published in 1903, most of the victims were boys, and most of the deaths resulted from blank-cartridge explosions, causing wounds of the hand. Contrast this with the tables of the "Surgical History of the War of the Rebellion," which show only 337 deaths from tetanus out of an enormous total of deaths from all causes.

Many cases of tetanus follow injuries received from rusty nails.

How does the bacillus cause the disease? When the bacillus is grown in bouillon it produces an intense poison or toxin, and experiments have shown that when this toxin is injected into susceptible animals it produces all of the symptoms of the disease. Vaillard, Vincent, and Rouget* have found that, as a rule, when the spores of the tetanus bacillus are introduced into the tissues, they are destroyed by the leucocytes, but when some of the tetanus toxin is introduced at the same time the spores develop. Schutze has shown that when a sterile putrefactive solution is added to an absolutely inactive solution of tetanus toxin, it becomes active and will produce tetanus.

The bacilli multiply at the seat of infection and there produce their toxins. The bacilli themselves have been demonstrated in the circulating blood as well as in the sheaths of the nerves leading from the wound and in the spinal cord. It is generally accepted, however, that most of the toxins are produced at the primary focus of infection. The question of the transmission of the toxins to the central nervous system has been the subject of extensive research, and has an important bearing on the treatment. The toxins have been repeatedly demonstrated in the circulating blood, the spinal cord and medulla, and in the cerebro-spinal fluid.

*Elting: Albany Medical Annals, Jan., 1904.

Morax, Marie, Meyer, and Ransom have shown that the toxins may be transmitted along the axones of the peripheral nerves to the central nervous system. From exhaustive researches Stintzing has concluded that the transmission may take place by means of the general circulation, but, as a rule, the toxins travel along the nearest nerves, and, upon reaching the spinal cord, produce, first, local tetanus, and then the toxins, becoming diffused throughout the cord and medulla, produce general tetanus. Elting has pointed out that when the toxins are transmitted along the nerves they find entrance into the spinal cord at the points of exit of the nerves and affect, first, the anterior-horn cells. However, it has not as yet been conclusively proven that the general circulation does not play an important part in conveying the toxins to the central nervous system, and therefore it must still be considered an important factor in the etiology of the disease.

Senn has called attention to the resemblance between the muscular spasms caused by strychnine poisoning and those due to the toxins of tetanus. He believes that if this and other drugs can act upon the spinal cord in such a manner as to cause spasms and muscular rigidity, we should expect that, if the microbe of tetanus produces toxins in the tissues, these latter might produce the same effect upon the cord, and that the symptoms are produced by them and not by the direct action of the microbe.

Pathology.—Aside from the presence of the bacilli and the toxins, no constant pathological changes take place, excepting those which are seen in the anterior-horn cells of the cord. The local wound may show a mild grade of inflammation. When a mixed infection is present the degree of inflammation seems to be due to the activity of micro-organisms other than the tetanus bacillus. There are no characteristic lesions of the nerve trunks and nerve centres other than those due to an irritant poison. Minute hemorrhages, dilatation of the capillaries, perivascular exudation, and other degenerative changes of the nerve cells have been described. Hyperæmia of the spinal cord and medulla oblongata frequently occurs. In the cord the anterior-horn cells are most constantly affected and show degenerative changes.

Symptoms.—The period of incubation is generally accepted to be from eight to fourteen days. Occasionally it is much shorter, and it may extend over three or four weeks.

In typical cases the symptoms usually make their appearance in a very gradual manner. There is, as a rule, the history of an injury, which may be very slight or may be extensive. Often the patient is presented with a history of having stepped on a rusty nail, or of having run a splinter into the foot, or of having received an injury from a toy pistol. In a little over a week, after a comfortable night's rest, the patient notices a slight difficulty in masticating his food or in opening his mouth. Some authors lay much stress upon the occurrence of local pain and slight or marked spasms of the muscles in the region of the wound, before the appearance of any jaw symptoms. It is a fact that this

so-called local tetanus occurs in all cases of experimental tetanus. Cases have been observed in which this takes place also in man, but, as a rule, the patients come to the clinician only after the more advanced symptoms have made their appearance. The occurrence of local tetanus adds weight to the theory of the transmission of the toxins along the motor nerves. If this mode of transmission is the true one, the local spasms and contractions of the muscles would be explained, the trismus and general spasms appearing when the toxins have been conveyed in sufficient quantities and of proper concentration to the medulla and spinal cord by means of the general circulation.

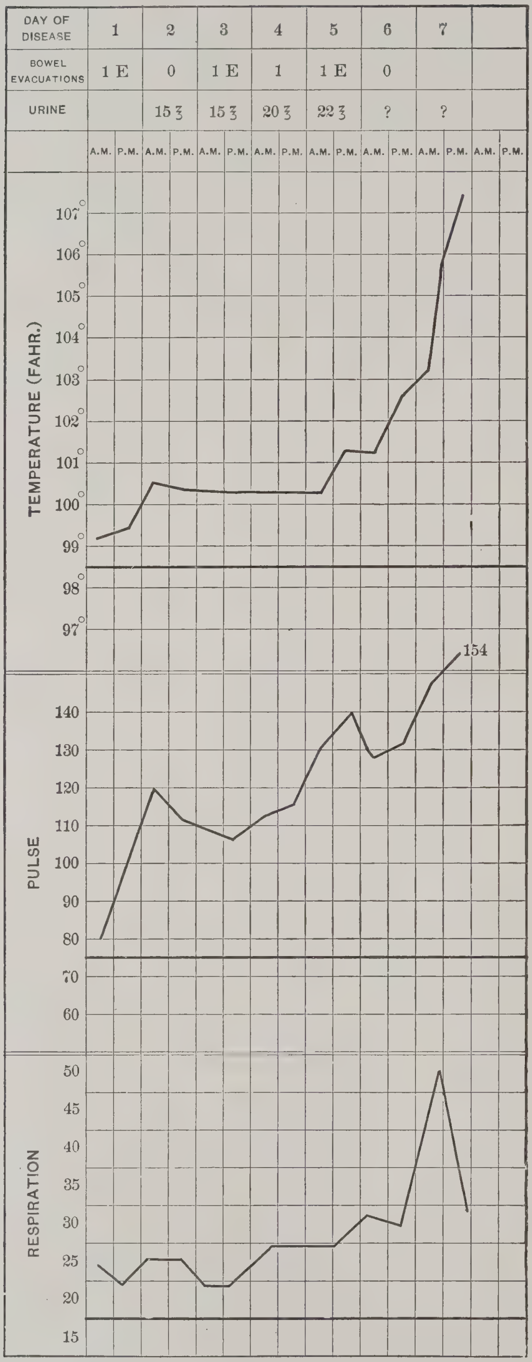


Fig. 135.—Chart Showing the Temperature, Pulse, and Respiratory Curves in a Typical Case of Tetanus.

The first symptom complained of is usually that of difficulty in opening the mouth. As the day advances this stiffness of the jaw increases, and is associated with pain in the cheeks or in the region of the temporo-maxillary articulation. Motion of the jaw greatly increases the pain. An examination of the muscles of mastication will reveal a rigidity and prominence of the masseter muscles. There is no soreness or swelling of the gums. When the patient attempts to bend the head forward, some resistance will be noted in the muscles at the back of the neck. The rotary muscles of the head are not affected. At the same time there is more or less difficulty in swallowing. The general condition of the patient is normal. He evinces no anxiety as to his

condition. As the disease advances the contraction of the masseter muscles becomes more marked, and the question of nutrition becomes a serious one. Attempts to feed the patient by mouth may increase the painful spasm of the muscles. The face may assume a peculiar expression, the "risus sardonicus," due to spasms of the facial muscles. But involvement of the orbicularis oris and other muscles of expression is not always present.

Already on the second day the jaws may be firmly locked. The stiffness of the neck muscles increases markedly, and, in consequence of the spasm of these muscles, the head will be drawn backward. The patients generally have a good appetite, but cannot eat. They complain of precordial pain and some gastric distress. Gradually, but surely, the abdominal muscles become involved. Both recti muscles are equally contracted, and are as hard as iron. The spasms are often accompanied by intense crampy pain, from which there is little relief. The patients have difficulty in emptying the bladder and rectum. The muscles are in a state of tonic contraction. As time advances, the stiffness of the neck becomes intensified and spreads to the muscles of the back. Sudden noises, or attempts at feeding, or the slightest irritation, may bring on paroxysmal clonic spasms affecting different groups of muscles. The most characteristic is that which affects the muscles of the back, producing opisthotonus, in which the spasm may be so severe that the body forms a half-circle, as inflexible as iron, being supported on the occiput and heels. Or the anterior pectoral and abdominal muscles may be affected, causing emprosthotonus. The pain at such times is always increased and the patients suffer excruciatingly.

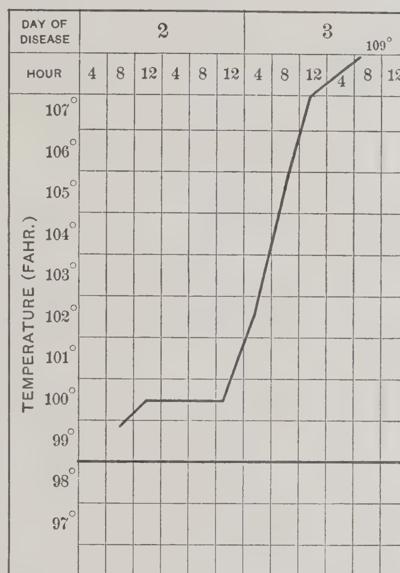


Fig. 136.—Temperature Curve from a Case of Very Acute Tetanus, with Rapidly Fatal Issue.

Varying degrees of muscular spasm may be present, at times involving only certain groups of muscles.

The mind is, as a rule, perfectly clear, and there is no impairment of the special senses.

In the most acute cases the advance of the disease is very rapid, and death may occur in from one to five days.

The temperature curve throughout the disease is variable and not characteristic. As a rule, there is a slight rise of the body temperature, which does not vary much during the twenty-four hours. As the end approaches there is usually a steady increase in the temperature, and frequently just before death

there is hyperpyrexia, the temperature reaching 107°, 108° F., and even higher (Fig. 136). In many cases there is a marked post-mortem rise, and cases have been reported in which it has reached 113° F. In the very acute cases hyperpyrexia is frequently seen (Fig. 135). In other cases, usually of the subacute type, the temperature may remain normal throughout. The pulse varies with the temperature, excepting in the afebrile cases. It is usually increased in frequency, averaging 110 to 120 to the minute. The respiration varies with the amount of involvement of the respiratory muscles.

Rapid emaciation and loss of strength are constant symptoms. As death approaches the pulse becomes rapid and irregular, and the end may come during one of the convulsive attacks, from asphyxia or cardiac dilatation, or the

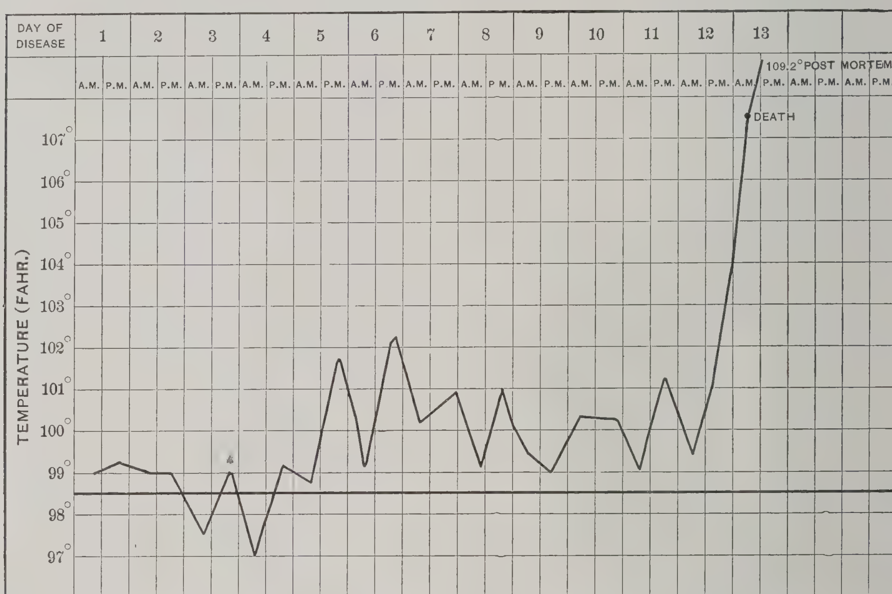


Fig. 137.—Temperature Curve of a Case of Tetanus, Showing the Marked Ante-mortem and Post-mortem Rises in Temperature.

patient may die of exhaustion. The disease may prove fatal in from twenty-four to forty-eight hours, or the suffering may be prolonged for six or seven days.

In the cases of *chronic tetanus,* the onset of the disease is more gradual and the symptoms are, as a rule, not so severe. The incubation period may last for from two to four weeks. The most constant symptom is the trismus, which usually is not so marked as in the acute cases, and allows of the feeding of the patient. The symptoms, however, may develop as rapidly as in the acute cases, but at no time are they as severe, and they may extend over a period of from six to ten weeks. Prostration is marked and the patients usually die from marasmus and exhaustion.

Tetanus neonatorum and *tetanus puerperalis* are really subdivisions of tetanus traumaticus, the symptoms of which have already been described.

Tetanus facialis, hydrophobicus, or head tetanus, is a special form of tetanus, following injuries to the head, especially wounds of the face. It is characterized by paralysis of the facial nerve on the side of the lesion, and later by trismus and spasms of the muscles of deglutition. Schultz has reported a very interesting case of this nature, in which the disease was caused by the bite of a peacock upon the forehead of the patient. Four days after the injury the first symptoms developed, and consisted of a paralysis of the left half of the face. On the eighth day after the injury the tetanic symptoms developed, and were confined to the muscles of the right side of the face and those of the head and neck. Recovery took place. Tetanus bacilli were demonstrated in the tissues of the wound. Most of these patients recover.

Diagnosis.—As a rule, the diagnosis is easily made. In many cases the bacillus of tetanus may be isolated from the wound. The cardinal symptoms are the history of the injury, the period of incubation, the spasm of the masseter muscles, and the rigidity of the neck. It may be differentiated from *strychnine poisoning* by the history of the case, the lack of tonic contraction of the jaw muscles, the rapid occurrence of clonic spasms, and the complete relaxation of the muscles between the paroxysms. If hydrophobia is suspected, the history of the original wound is important. In *hydrophobia* the incubation period is longer, and there are present: spasmodic dysphagia, dyspnœa, an absence of trismus and opisthotonos (Butler), and more mental disturbance. In *tetany* the nature of the spasm is different, and it usually is limited to the extremities. Trismus rarely occurs.

Prognosis.—The prognosis is always grave, but, since the introduction of antitetanic serum, treatment of the disease has shown more favorable results. Moschcowitz* maintains that the prognosis depends upon two things:

(1) The period of incubation. In a general way, the shorter the period of incubation the worse is the prognosis. This, however, is dependent on the second factor:

(2) The rapidity of the development of the symptoms and their severity.

Puerperal tetanus and tetanus neonatorum are almost always fatal. In tetanus facialis and in those cases in which the spasm remains localized in the head and neck muscles, the prognosis is more favorable.

Treatment.—Of greatest importance is the prophylactic treatment of tetanus. In many cases such treatment is not possible, but when it is remembered that more than half the cases which develop in the United States are due to wounds inflicted by a rusty nail and Fourth-of-July casualties, it would seem that too great care could not be exercised in the management of these patients. In such wounds, and in wounds in which dirt has been ground into the tissues, especially in barnyard injuries and in those which have been inflicted in localities where tetanus is endemic, the strictest antiseptic precautions should be adopted and the wound treated openly. It should be the invariable rule

* Annals of Surgery, vol. xxxii., p. 231.

that in such cases the patient shall receive an injection of 20 c.c. or more of an approved antitetanic serum. This rule has been applied in many instances and with the most brilliant results. It is the writer's opinion that more patients have been prevented in this way from acquiring the disease than have been cured by the use of the serum when the disease had become well established.

When the patient is presented with well-marked or beginning symptoms of tetanus, the treatment should be carried out in a routine way. The indications are: 1st, to limit the local activity of the tetanus bacillus; 2d, to neutralize the toxins which have been produced; 3d, to eliminate the toxins as far as possible; 4th, to nourish the patient; and 5th, to control the convulsions.

Local Treatment.—In small wounds it may be possible to excise the entire area of pathological tissue. However, in most cases this is not possible, and the wound should be laid open, all foreign material and necrotic tissue should be removed, and the parts should be thoroughly cleaned with some strong antiseptic solution. Probably the two best solutions to be employed in such cases are carbolic acid and tincture of iodine, and they should be freely used. Some writers recommend the employment of the thermocautery. The question of amputation of the member which is the seat of the infection must be decided by the individual surgeon. Some favor the operation, especially where there has been extensive injury to a part or where suppuration is present; others believe that it is useless, holding that the toxins are already present in the tissues and circulating blood in sufficient quantities to cause death, and that consequently the operation would do more harm than good.

Tetanus Antitoxin.—The antitoxin has no bactericidal action. It protects only against the tetanus toxin. It is to be regretted that no standard of strength has as yet been obtained in the production of the tetanus antitoxin, and therefore the dose must vary with the preparation. It may be injected subcutaneously or intravenously, and, in the urgent cases, directly into the brain, or by means of a lumbar puncture subdurally. Doses of from 5 c.c. to 150 c.c. of the serum have been injected without apparent injury to the patient. As a routine procedure it is well to inject small doses, from 10 to 20 c.c. of the serum, subcutaneously into the loose cellular tissue of the back or abdomen, and repeat it two or three times a day. In the more severe cases lumbar puncture may be done, between the third and fourth lumbar vertebræ, and, after allowing from 10 to 15 c.c. of cerebro-spinal fluid to escape, a like quantity of the serum is injected. Kocher has recommended injecting 10 c.c. of the serum into the lateral ventricles of the brain. This may be repeated every day and combined with subcutaneous and intravascular injections. Some authors recommend that very large doses of the serum (200 to 300 c.c.) be injected during the twenty-four hours. The most important thing to emphasize is the necessity of injecting the serum as soon as the disease is suspected or recognized.

If we accept the theory of the transmission of the toxins along the motor nerves to the spinal cord as the true one, it would naturally follow that an injection of serum directly into the membranes of the cord, at the same time slightly abrading the nervous tissue, would be apt to be followed by more rapid results, in that the serum would more rapidly come into contact with the toxins affecting the anterior-horn cells. It has furthermore been demonstrated, with a certain degree of certainty, that the toxins cannot travel in a peripheral direction along the motor nerves, and that it takes several days for the toxins to travel from the end of a nerve to its origin; also that antitoxin pursues exactly the same course. Accepting these theories, Rogers* has advocated a combined intraneural and intraspinal injection of serum, a procedure which he has carried out successfully in two cases. In the first case, in which tetanus followed an injury to the hand, he injected into each one of the nerves of the left brachial plexus five to ten minims of antitoxin. In addition to this, sixty minims were injected into the spinal cord. This was repeated the following day. In his second case, following an injury to the foot, he exposed the anterior crural and sciatic nerves on the right side and injected into each half a drachm of antitoxin, combined with a spinal injection of one drachm and a half of serum. The spinal injection was repeated on three successive days. Both patients recovered.

Carbolic-acid Treatment.—This method of treatment may be used independently or combined with the serum and drug treatment. It was first advocated by Bacelli, who noted that injections of carbolic acid were followed by a marked diminution of the reflex excitability of the nervous system, and he has ascribed to it certain general antiseptic properties. The amount of the solution which should be injected varies according to different experimenters. Kocher recommends the systematic subcutaneous injection of carbolic acid, in doses of ♏xv. of a three-per-cent solution every one or two hours, according to the severity of the case. According to Elting (*loc. cit.*), Ascoli in one case injected 72 c.c. a day for seven days, and 60 c.c. a day for forty-one days. Symptoms of carbolic-acid poisoning have never been observed in cases so treated. It should be used as a routine treatment in all severe cases.

Drug Treatment.—Drugs are indicated to control the symptoms, especially for the prevention and relief of spasms. Opiates may be administered freely. Morphine should be given by hypodermic injections, alternating with large doses of chloral hydrate and the bromides given per rectum. To control the more severe spasms chloroform should be given temporarily, especially where there is much dyspnœa. Amyl-nitrite pearls may be called for in spasm of the glottis.

To sustain the patient and contribute to the circulating fluids, as well as to dilute the toxins and aid in their elimination, subcutaneous infusions of decinormal salt solution should be given.

Nourishment.—From the onset of the symptoms this becomes an important

* Annals of Surgery, vol. xl., pp. 417, 759.

question, and systematic and regular feeding should be prescribed. In the milder cases small amounts of highly nutritious food may be given by the mouth. When, however, it is impossible to administer food in this manner, or when the act of swallowing becomes too painful or impossible, regular nutrient enemata should be given per rectum, supplemented by subcutaneous injections of ℥iij.–vi. of oil, as suggested by Leube.

In the general management of the case, careful nursing and absolute quiet are essential. The patient should be isolated in a dark room, as far removed as possible from any noise. Sudden draughts of air and many seemingly slight external stimuli tend to produce spasms.

SURGICAL SHOCK.

BY JOSEPH C. BLOODGOOD, M.D., Baltimore, Maryland.

THIS subject is one of surgical physiology. At the present time the exact etiological factors are not definitely and conclusively proven. It is by no means a simple problem to investigate, either experimentally in the physiological laboratory or clinically in surgical practice.

For practical purposes, shock should be considered a condition of general depression produced by various causes. These factors act through the medium of afferent nerves upon various centres in the spinal cord and brain, especially the vasomotor centres. Howell, from his physiological experiments, recognizes a cardiac shock as well as a vasomotor shock. It is a question whether the sympathetic ganglia are also deleteriously influenced by the various factors which may produce shock.

In this condition, which we call shock, there is always a fall in the blood pressure, but a fall in the blood pressure alone should not be considered a positive indication of shock. All of the nerve centres react feebly to afferent stimuli, the pulse is usually rapid and feeble, the respiration shallow, the pupils somewhat dilated, reacting feebly to light; all of the cutaneous reflexes are lessened; there is increased perspiration, for which reason the skin feels cold and moist; the temperature of the body is lowered; the mental condition is usually one of quiet depression, if I may use this term; there is at first no delirium, nor delusions. All the mental faculties are less acute, but the patient, nevertheless, is perfectly conscious. There is no pain or discomfort, but the patient realizes his depressed condition and will inform you that he feels weak.

At the present time it is impossible to define shock, chiefly because its exact physiology has not been completely worked out. For this reason we must use the term in a composite sense for a clinical picture which varies in degree, and is produced by various factors, some of which we understand and can demonstrate, many of which apparently we do not understand. Future experimental physiological work and more exact clinical observation with instruments of precision may simplify the question and allow us to classify shock according to the exact factor or factors which produce it. We may also be able to define and recognize different clinical pictures in relation to different etiological factors.

For this reason a discussion of the subject must be limited. One can only present, first, the results of physiological experimental work which are suf-

ficiently accurate to allow conclusions for practical purposes in the clinical recognition and treatment of shock, and to discuss the question from a purely practical and clinical standpoint only so far as we are quite positive of the accuracy of our observations and experience.

In man and animals after death from shock the gross and microscopic changes, with our present means of estimating them, are practically *nil*. One finds that the blood is accumulated chiefly in the veins, especially of the splanchnic system.

THE PHYSIOLOGY OF SHOCK.

Schaefer ("Text-book of Physiology," 1900, vol. ii., p. 845) states that shock, like collapse, is a term more used by the clinician than the physiologist, and that it is a term somewhat ill defined in its scope. By the physiologist "shock" is understood to be primarily a nervous condition. "If in a frog the spinal marrow be divided just behind the occiput, there are for a very short time no diastaltic actions in the extremities; the diastaltic actions speedily return; this phenomenon is shock." By diastaltic action is meant the muscular movements of the extremities which take place after stimuli. Shock may be considered, according to the physiologist, the whole of that depression or suppression of the nervous function which ensues forthwith upon a mechanical injury of some part of the nervous system and is of a temporary nature (Schaefer). Physiologists, therefore, are somewhat uncertain whether the phenomenon of shock should be considered one of depression or of inhibition. In animals, spinal shock is a distinct phenomenon. One makes a section of the spinal cord, the spinal reflexes disappear, but to reappear, although the spinal cord be still divided. That is, the mechanical injury produced in dividing the spinal cord affects temporarily the centres below in such a way that for a certain time they do not react to stimuli, yet these centres themselves have received no mechanical traumatism. My careful reading of the physiologists' description of spinal shock has given me a better conception of surgical shock, which may be correct. Surgical shock is associated with mechanical injuries in various parts of the body, near or remote from various nervous centres; these centres are affected by afferent stimuli, either of a depressing or of an inhibitory nature; the result is that these centres do not react at all, or react less promptly for a certain period of time. In some instances the effect on the centre is sufficient to prevent it from reacting for such a length of time that, if it is one of the so-called vital centres in the medulla, death takes place.

It has been my good fortune to be able to discuss the physiological problems of shock with Dr. Howell, professor of physiology of the Johns Hopkins University, and to read the original manuscript of a paper entitled "An Experimental Study of the Cause of Shock," which was written quite recently. The chief virtue, therefore, of my remarks on the physiology of this subject is due

to the source of my information. I have had no practical experience in the physiological laboratory. According to Howell there is some uncertainty as to the immediate physiological cause of surgical shock. In the human individual the mental and physical symptoms of shock would indicate a marked depression of the activity in the nerve centres, and not infrequently these symptoms increase in severity and terminate in death.

In observations on lower animals, this condition is associated not only with a faint and rapid heart-beat, but with a pronounced and permanent fall in the blood pressure.

The relation of a fall in the blood pressure to shock is such an important one that I will give Howell's remarks in detail and *verbatim*, rather than attempt to summarize:

"Since this latter symptom (*the fall in blood pressure*) is sufficient in itself to explain many, if not all, of the other features exhibited in shock, it has been seized upon as the really significant and causative factor. The condition of an animal in extreme shock is in fact similar to that of an animal whose vasomotor centre has been destroyed or has been severed from its connections with the blood-vessels. Under both conditions the complete paralysis of the blood-vessels results in a fall of the blood pressure to 40 to 20 mm. of mercury and a rapid, feeble heart-beat; and unless in some way the vascular tone is restored or the force of the heart-beat is increased, the circulation soon becomes more and more feeble and finally ceases. While the activities of the other nerve centres, particularly, as will be shown, that controlling the heart, may also be depressed primarily in shock, the failure of the vasomotor centre with the subsequent vascular dilatation and fall of blood pressure, seemed to be sufficient in itself to lead to a fatal termination, and this symptom may, therefore, be regarded as the most essential and most dangerous feature of shock. In surgical experience and in experimental observations it is noticed that what we call shock may exist in very different degrees of severity. It may be light and comparatively transient, or severe and fatal, or severe with rapid or slow recovery. How the blood pressure varies under the different conditions in man has not as yet been determined with accuracy, but in experiments on lower animals it seems quite certain that permanent recovery, when it occurs, follows upon the restoration of blood pressure to normal limits. It would seem justifiable, therefore, to connect the varying degrees of severity of shock with the duration and extent of the vascular paralysis. Vascular dilatation, however, is a normal physiological occurrence, and, under strictly functional conditions, it may be sufficiently extensive to cause a distinct fall in blood pressure; while under experimental conditions, as every physiologist knows, we may in various ways obtain a very marked fall in blood pressure in consequence of vascular dilatation. Stimulation of depressor-nerve fibres, in the depressor nerve from the heart, for instance, or in the nerves from other regions, such

as the ear or testes, will bring about such a vascular dilatation. We do not, of course, speak of a fall of pressure produced in this way as a condition of shock. The effect passes off promptly as soon as the stimulus ceases.

"When Crile, therefore, in his recent work on surgical shock, makes the statement that 'the essential phenomenon in shock is a diminution of blood pressure,' his words, unless properly qualified, are erroneous and misleading. To call every diminution in pressure a condition of shock amounts to giving a new meaning to the term and is tantamount to saying that shock is a constant and normal occurrence in the functional activity of the body. We have reason to believe that a state of syncope may be due to a sudden and large fall of blood pressure, but even such an extreme and abnormal variation is not properly regarded as shock, since recovery is, as a rule, prompt and complete. If, therefore, we say that the most important symptom of shock is a more or less complete paralysis of vascular tone, we must set certain limits to the extent and especially to the duration of this paralysis, and designate the condition as one of shock only when it passes beyond these limits. Under experimental conditions we recognize the fact that in extreme shock the vascular paralysis is as absolute as it would be were every vaso-constrictor fibre in the body divided or the vaso-constrictor centre destroyed. Between this condition and that of functional vascular dilatation with slight fall of blood pressure we may presumably have every degree of variation, but we are not justified in using the term shock except in those cases in which the vascular paralysis is not only extensive, but is maintained for a long period after the original stimulus causing it has ceased to act. It should be added, perhaps, that in what precedes I have not taken into consideration the shock that may be produced solely by severe hemorrhage. That a great loss of blood should bring about a fall in blood-pressure and a diminution in the activity of the heart and central nervous system is, so to speak, self-evident and needs no attempt at explanation. We are concerned here only with those conditions in shock in which the hemorrhage alone is not sufficient to explain the result, and can only be regarded as an adjunct factor. If we grant the essential correctness of the general statements made above, it is evident that two main problems confront us. First, what is the immediate cause of the profound and prolonged vascular paralysis, and, second, what means may be used to restore the blood-vessels to their normal tone? Experiments that I have made recently throw, I believe, some light upon these problems, and have led me to take a point of view somewhat different from that usually assumed in discussing the causation of shock. The experiments were made upon dogs, and shock was produced by one of the following methods: 1. *Exposure and handling of the abdominal viscera;* 2. *Long-continued stimulation of the cutaneous nerves, effected usually by the application of hot-water bags to the skin;* 3. *By operations on the brain involving removal of a portion of the skull and dura, and in some cases also*

of the cerebrum. It should be added that in all cases the dogs were completely anæsthetized with morphia and ether. Whether anæsthetization with ether aids or retards the production of shock cannot be stated definitely, but in human beings shock from operations under ether occurs often enough, and in dogs under ether the vasomotor reflexes at least would indicate a favorable condition for shock. However this may be, it was my experience, as it has been that of other observers, that the production of shock in dogs is a very uncertain matter. In some cases it comes on after comparatively little operative violence, while in other cases prolonged operations, wide exposure of the abdominal viscera, fall of body temperature, and some hemorrhage might fail to induce shock. In such negative cases the vasomotor reflexes, as will be explained presently, indicate that the animal was, so to speak, approaching a condition of shock.

"Of the several methods that I used the one that caused shock most promptly and most frequently was operation on the brain. In some cases mere exposure of the brain produced complete shock, while in other cases, after removing a portion of the skull, the animal still showed a normal pressure, but fell into shock more or less rapidly after ablation of portions of the cerebrum. The animals were, in all cases, connected with the kymograph, and records were taken continuously of the blood pressure and respiration. Some of the details of these experiments have been published already "Contributions to Medical Research' dedicated to Victor C. Vaughan), and it may suffice to give here a summary of these results and my conclusions:

Howell's Conclusions on Shock.—" 1. Shock is characterized by a long-continued, low arterial pressure (vascular shock) due to partial or complete loss of activity of the vaso-constrictor centre, and by a rapid, feeble heart-beat (cardiac shock) due in part, at least, to a partial or complete loss of activity of the cardio-inhibitory centre.

" 2. Cardiac shock may occur more or less independently of vascular shock, but vascular shock is always preceded or accompanied by cardiac shock. The respirations in shock are diminished in amplitude and usually in rate.

" 3. Shock may be produced experimentally by severe operations of various kinds, but most often by extensive operations on the brain.

" 4. The physiological evidence in experimental shock indicates that the condition is due fundamentally to a strong inhibition of the medullary centres (vaso-constrictor, cardio-inhibitory), leading to a long-continued suspension of activity, partial or complete.

" 5. Injections of alkaline solutions of sodium carbonate intravenously or into the rectum during shock increase markedly the amplitude of the heart-beat and bring about a rise of arterial pressure. When the shock is moderate (aortic tension 60 to 70 mm. Hg) the injections may restore arterial pressure to an approximately normal level. When the shock is severe (aortic tension

of 20 to 40 mm. Hg) the injections may increase arterial pressure by about 100 per cent for long intervals, and the effect, when it wears off, may be restored by repeating the injections. The effect of the injections is due chiefly or entirely to a direct action on the heart.

"6. Stimulation of sensory nerve trunks or sensory surfaces in an animal in a condition of shock leads to a further fall of pressure, and to this extent augments the condition of shock.

"7. The blood of animals in a condition of shock has no toxic action when injected into the circulation of a normal animal."

Blood Pressure.—At the present time our only accurate method of estimating shock, either in the physiological experiment or in surgical practice, is by means of an apparatus which records the blood pressure. This is not the place to discuss the relative values of the different contrivances used for this purpose. Howell and other physiologists are of the opinion that it is of the greatest importance to employ an apparatus which records both the diastolic and systolic pulse waves. Unfortunately, at the present time there is neither in the literature nor in the experience of the surgical clinic with which I am connected a sufficient number of these accurate blood-pressure records in surgical cases to allow any positive conclusions as to their practical value. I am of the opinion, however, that this instrument of precision should be employed more frequently in surgical cases, and I would suggest to those interested in this subject to read the contributions of Erlanger and Hooker published in the Johns Hopkins Hospital *Reports* (vol. xii., 1904), entitled "An Experimental Study of Blood-Pressure and Pulse-Pressure in Man," and the literature which I have discussed in *Progressive Medicine* for December, 1903, and December, 1905.

Physiological Experiments to Estimate the Effect of Various Manipulations, During Operation, on Blood Pressure, and Their Relation to Shock.—The practical surgeon should have knowledge of the following data: The effect of the general narcotic on blood pressure and its relation to shock; whether in certain operations shock is less if the operation is carried on under spinal or local anæsthesia; the effect of hemorrhage, the duration of the operation, exposure of tissues to the air, effects of extreme heat or cold, the effect of manipulation or injury on various tissues or organs.

For these data Crile, of Cleveland, has without doubt published the most important contributions, and I shall draw largely upon his two works: "Surgical Shock" published by the Lippincott Company in 1899, and "Blood-Pressure in Surgery" published by the same firm in 1903.

We may summarize the results of this experimental work as follows: Hemorrhage is one of the most important factors in the production of shock. As it is practically under the control of the surgeon, this element can in the majority of cases be eliminated by painstaking hæmostasis. A long bloodless

operation is much less serious than a short and bloody one. A general anæsthetic must be considered in all cases a factor. The significance, however, of this factor varies. Chloroform is always associated with a fall in blood pressure, ether with a rise in blood pressure.

Gentle manipulation and incision of tissues with a sharp knife affect the blood pressure less than rough manipulation and tearing of tissues. The greater the number of afferent sensory nerves in the tissue manipulated, the greater the effect on the vasomotor centres. These experimental findings are well borne out by clinical observations. During an operation, therefore, the amount of the anæsthetic should be as small as possible; the operation should be performed as quickly as is compatible with the safety of the patient and the purpose of the intervention; tissues should be handled as gently as possible; if large nerves must be divided, as in amputations, they should be blocked with cocaine injection; tissues should be exposed to the air only when absolutely necessary; they should then be protected with warm moist gauze; the patient should not be exposed to extreme degrees of heat or cold.

The weaker the patient to be subjected to operation, the more attention must be given to these details which lessen shock.

Skin.—Simple incision of the skin has practically no effect upon blood pressure; burning of the skin causes a uniform rise in the pressure. The effect is more marked in animals when the skin of the paws is burnt. All Crile's observations tend to show that the greater the nerve supply of the area of skin the greater the shock when this area is subjected to injury. The relation of extensive skin injuries to shock is well illustrated in burns. The wider the area of a skin burn the greater the shock; the depth of the burn is not a factor. In a superficial burn there is just as much injury to nerve ends as in the deep burn. Undoubtedly in burns there is another factor in the cause of death, besides the tremendous effect on the vasomotor centres from the extensive peripheral irritation—that is, the effect of the toxins undoubtedly produced in the burnt tissue.

Pain in the normal individual produces a rise in the blood pressure; but when we are dealing with weakened organisms or a patient already shocked, pain is an afferent impulse which has a decided depressing effect on the medullary centres. For this reason, in the treatment of such patients everything should be done to prevent, lessen, or block it. In such cases a skin incision without an anæsthetic becomes an element which increases shock, and the danger of the anæsthetic is less than the danger of the painful incisions or manipulation. It requires a deep narcosis with a general anæsthetic to inhibit the painful effect of a skin incision. For this reason, when on account of the weakened condition of the patient, or in the presence of shock, one does not wish to produce a deep general narcosis, the operation should be performed under a combination of local and general anæsthesia. The skin incision can be

made perfectly well under the infiltration method of Schleich, the deeper incision by peri- or intraneural injections; further manipulations under general narcoses.

This demonstrates how important it is for the surgeon to be familiar with the sensitiveness of different tissues. This knowledge is not only of value when performing operations under local anæsthesia, but in preventing shock by anæsthetizing with a local or general anæsthetic the sensitive tissues. Lennander's publications on this subject (*Mittheilungen a. d. Grenzgeb. der Chir. und Med.*, 1902, vol. x., and *Deutsche Zeitschr. f. Chir.*, 1904, vol. lxxiii., p. 297, and *Mittheilungen a. d. Grenzgeb. d. Chir. u. Med.*, 1906, vol. xv.) are the most recent and the best.

Connective Tissue.—Crile was unable to find any effect of injury to this tissue, providing nerves were not included.

Muscle.—The effect of tearing and crushing of muscles was attended by practically the same phenomena as those observed from similar manipulations of the skin, but to a very much lesser degree. Lennander did not investigate the sensibility of muscles. In my own experience with operations under local anæsthesia I found that the muscle is not sensitive. However, the nerve near or within the muscle is very sensitive. For this reason, in operations under local anæsthesia, one must be familiar with the position of the various nerves so that they may be rendered anæsthetic by neural or intraneural injections. In cases of amputation on patients suffering from shock I am inclined to the opinion that, if possible, one should avoid division through large muscle bellies; instead, he should select if possible the joint or a position where most of the muscles are tendinous.

Bone.—According to Lennander the marrow and the cortical bone are insensitive, while the periosteum is very sensitive. Crile's experimental work confirms this finding. I am of the opinion that the rapid sawing of a bone would be attended with very little, if any, shock. It is very easy, however, to render the periosteum insensible with cocaine infiltration. I have repeatedly resected one or more ribs painlessly after this method in patients in a very critical condition, with empyema or lung gangrene.

Joints.—According to Lennander's most recent communication (*Mittheilungen a. d. Grenzgebieten d. Med. u. Chir.*, 1906, vol. xv., p. 465) the joint capsule has sensory nerves, but the articular cartilage is insensible. Crile was unable to demonstrate any deleterious effects from various manipulations on large joints. Under local anæsthesia I have been able to open and irrigate without much pain most of the joints. Arthritis increases the sensitiveness of the surrounding tissues. Dislocations and compound injuries of the joints are not associated with any considerable degree of shock unless the soft parts and nerves in the neighborhood are injured.

Nerve Trunks.—Crile's experimental work demonstrated that the greatest effect upon blood pressure was produced by injuries of nerve trunks. A quick

severing with a sharp instrument had much less effect than crushing or tearing. The effect on the centres from injuries to nerve trunks can be entirely inhibited by a local infiltration of cocaine centrally to the point of injury. This laboratory experiment is confirmed by quite a number of clinical observations. In amputations, especially if the patient be in a condition of shock, the large nerve trunks should be blocked.

Operations upon the Head.—In operations upon the brain, incision of the scalp and the making of a bone flap are attended with little effect upon the blood pressure, but exposure and manipulation of the dura and the brain itself have a decided effect. This experimental finding is confirmed by clinical observation.

In operations upon the tongue, the floor of the mouth, and the lips, if there is no loss of blood, there is no effect upon the blood pressure. This experimental finding is also confirmed by clinical observation.

Injury of the mucous membrane of the nose had no especial effect upon the blood pressure, but in some cases there was a temporary and partial inhibition of respiration and the heart. Although the mucous membrane of the nose and the pharynx is quite sensitive, I have never been able to convince myself that during operations incision or injury of this tissue had any appreciable effect on blood pressure or was adding to shock, but I think that this needs further investigation, and I am inclined to the opinion that, in patients in whom we fear shock during operation, one should anæsthetize this very sensitive mucous membrane with cocaine.

The effect of injury of the mucous membrane of the pharynx, the soft palate, base of the tongue and epiglottis is one of inhibition on the heart and respiration to a certain extent.

Larynx, Trachea, and Œsophagus.—The chief effect of irritation of the mucous membrane of the larynx, even under anæsthesia, is similar to that of the epiglottis—a temporary arrest of respiration. If great force is used, there may be a partial or complete inhibition of the heart. The larynx, however, quickly becomes tolerant to this manipulation, and the effects are not noted. If the superior laryngeal nerves are divided, these manipulations produce no effect on the respiration and heart action. The effect of the application of cocaine was similar to the division of the superior laryngeal nerve. This experimental finding of Crile of the effect of cocaine used on the mucous membrane of the larynx is an important one to remember. I am inclined to the opinion that in extensive operations on the larynx, base of the tongue and epiglottis, and sensitive mucous membrane of nose and pharynx a preliminary application of cocaine would be beneficial in that it inhibits the afferent impulses which have an inhibitory effect on heart and respiratory action.

Good (*American Medicine,* August, 1902, p. 293) asked the question "are not some deaths during operation in regions supplied by the trifacial nerve, due to reflex inhibition of respiration and the heart?"

Tracheotomy.—When this operation is done under cocaine, no changes are observed in blood pressure. As a rule, when the trachea is first opened, its exposure to the air produces coughing, and there may be for a moment a slight asphyxia. This effect is less if the patient is under general narcosis.

Asphyxia.—According to Crile asphyxia produces a rise in blood pressure, to be followed, after the asphyxia has passed, by a fall in the pressure with an increased rapidity of pulse. Experimental investigation on the effect of asphyxia on the vasomotor centres is not complete. In practical surgery when asphyxia takes place during ether narcosis and the patient's general condition is good, the effect upon the patient's general condition as observed by the ordinary methods is not very marked. In weak patients, however, this asphyxia and cyanosis become very dangerous factors, and the anæsthetist should use every caution to prevent their occurrence. The asphyxia or cyanosis produced by the administration of nitrous oxide has an entirely different effect from that produced by an obstruction to breathing. In the various contributions on general anæsthesia, when this method has been used to introduce narcosis, no bad effects upon the blood pressure have been observed. I have been unable to find any important contribution with observations on the use of nitrous oxide in cases of shock.

Operations on the Neck.—Lennander has demonstrated that the thyroid gland itself is insensible. This has been observed by all surgeons who have performed thyroidectomies under local anæsthesia. Cocaine infiltration is only absolutely necessary for the skin incision. Sometimes in the division of muscles the patients complain of pain. Now and then when the thyroid vessels are ligated, especially the veins, the patient may complain of some discomfort, but the remainder of the dissection is carried on practically without pain. The patients complain of great discomfort, however, when traction is made. If the tumor is very adherent to the trachea, pain is experienced during this dissection. It is remarkable how little shock is observed in the extensive operation for the removal of larger goitres, providing no blood is lost. The few blood-pressure records confirm this. Even in patients with exophthalmic goitre who at the time of the operation may have a very rapid pulse, one observes no shock. Although the pulse is rapid in this disease, the blood pressure is high, and in the few records has been maintained during the thyroidectomy under cocaine infiltration. The chief danger is acute dilatation of the heart. The insensibility of a thyroidectomy and the absence of shock, if no blood is lost, are one of our best examples illustrating that extensive dissection, when made through insensible tissue, has little or no effect on the vasomotor centres. A contrast to this finding is seen when one attempts to excise a diffuse lipoma of the neck, or a mass of tuberculous or Hodgkin's glands. The dissection may cover a smaller area than a thyroidectomy, but this area contains numerous branches of cerebro-spinal nerves, difficult to expose and block with the

cocaine infiltration. These patients suffer more pain and quickly show symptoms of shock.

Extensive dissections of the neck are always attended with a certain amount of shock, even if they are bloodless. The degree of shock is in direct proportion to the condition of the patient. In weak patients, therefore, these extensive dissections become elements of danger. The shock undoubtedly is less if all manipulations are made with the greatest gentleness, when tissues are divided quickly with a sharp knife, and when blunt dissection is avoided. In operations of this character, I believe that the pneumatic rubber suit of Crile should be employed to maintain blood pressure. (See Figs. 140 and 141.) I will discuss this again under Treatment.

Operations on the Thorax.—These may be divided into two groups. In the first the thoracic cavity is not opened, in the second this cavity is opened. The most common operation on the chest belonging to the first group is the extensive dissection for carcinoma of the breast. Crile in his observations on blood pressure in these cases noted only moderate changes. Toward the end of the operation, especially during the dissection of the axilla, when large blood-vessels and nerves are manipulated and divided, a fall in the blood pressure is observed. Crile, however, especially notes that when the dissection is done with a sharp knife and with minimum traction the fall in blood pressure is insignificant. The change in blood pressure is noted to be marked when the tissues are handled roughly, when the dissection is blunt, when traction is made. These sentences of Crile sound the keynotes to the factors which on the one hand avoid, and on the other increase shock.

When our patients are strong, the result of the loss of a certain amount of blood, blunt dissection, rough handling, unnecessary traction and ligation of tissues *en masse*, although they would cause a change of the blood pressure, if such a record were kept, do not produce sufficient depression to be of much clinical significance. The patients are a little shocked, convalescence is a little prolonged. However, when our patients are very old or very young, or in a weakened condition from any cause, these rough manipulations become serious factors, so serious in some cases that the operation cannot be completed at all, or only in a very hurried and unsatisfactory manner.

In the very large experience in the surgical clinic of the Johns Hopkins Hospital with the extensive operation for carcinoma of the breast, I have been struck with two very significant facts—the absence of shock and the apparent low percentage of post-anæsthetic complications. The average anæsthetic time of this operation has been at least two hours, rarely less than an hour and a half, frequently two and a half hours, now and then three, and in a few cases four hours. The anæsthetist in this group of cases seems to be impressed with the fact that it is to be a long operation. For this reason he is unusually careful with the anæsthetic, which has always been ether. The

patients are seldom completely narcotized. Although the average age is high, I find that the post-operative pneumonia is distinctly less frequent per hundred than after operations for hernia. We have not many blood-pressure records; but, as far as clinical observations can record, shock is rarely observed, and has never been serious. This apparently has been due to the method of dissection established by Halsted. From the skin incision, throughout the operation, every bleeding point is clamped; the dissection is made slowly and carefully; in the axilla especially, vessels and nerves are handled with the greatest gentleness, they are isolated and ligated separately; nerves are cut quickly; blunt dissection is never employed, except for loose fat and cobweb connective tissue devoid of nerves.

Crile records that resection of the ribs caused but slight change of the blood pressure, but opening the pleural cavity a marked change. When an empyema is drained the opening of the cavity and the discharge of pus are attended with a rapid fall of blood pressure and an increase in the pulse rate. Here we have another excellent and concrete example of the elements which produce shock and affect blood pressure. According to Lennander the parietal pleura, like the parietal peritoneum, is extremely sensitive. Manipulations of either are almost impossible under local anæsthesia. The lung itself is insensitive, and the probabilities are that even extensive manipulations and cutting of the lung would of themselves be devoid of any effect on the vasomotor centres. It is the opening of the pleural cavity that is the chief factor of danger in all intrathoracic operations, chiefly owing to the change of atmospheric pressure with its primary effect upon respiration and secondary effect upon the blood-pressure. The observations of Crile on operations upon empyema are important to recollect. In weak patients pleural effusions should be evacuated very slowly. The dangerous effects of opening the pleural cavity due to collapse of the lung and its effect upon respiration and circulation have limited this field of surgery. This is not the place to discuss Sauerbruch's experimental work or his pneumatic operating chamber designed to equalize atmospheric pressure and eliminate the dangers of intrathoracic operations, nor to consider Brauer's method designed for the same purpose. I have discussed these contributions in the *International Clinics* for April, 1905, p. 300.

Extensive resection of ribs, on account of injury to the intercostal nerves, even if the pleural cavity is not opened, and no blood is lost, is an operation attended with some shock. From a limited experience I am inclined to the view that even if general narcosis is employed the intercostal nerves should be blocked by a perineural injection of a weak solution of cocaine. The nerve lies in the groove beneath the rib. In operations on the thorax the sensitive tissues are chiefly the skin, the periosteum of the rib, the intercostal nerves, and the parietal pleura.

Diaphragm.—According to Lennander the parietal peritoneum and the pleura on the diaphragm are unusually sensitive. Crile has demonstrated that manipulations of the diaphragm, even though slight, have a distinct effect upon respiration.

Undoubtedly in all intrathoracic and abdominal operations manipulations of and traction on the diaphragm should be avoided if possible.

Abdomen.—Lennander's observations on the sensitiveness of the peritoneum and abdominal viscera are the most exhaustive extant. The parietal peritoneum is extremely sensitive. The visceral peritoneum and the viscera themselves are insensitive, that is, all tissues innervated from the sympathicus and lower vagi are insensitive to touch, pain, and temperature. The reverse is true of all tissues supplied by branches of the cerebro-spinal system. These observations are important not only for the technique of local anæsthesia, but also in operations upon the abdomen, when we wish to lessen or avoid shock by reducing to a minimum sensory impulses in cerebro-spinal nerves. At the present time there are no data, experimental or clinical, to indicate that manipulations of any kind on these insensible viscera or tissues have any deleterious effects on the centres of circulation and respiration. Handling, tearing, and cutting the abdominal viscera are not harmful if done without loss of blood. We must recollect, however, that traction on these viscera irritates the posterior parietal peritoneum and the connective tissues containing nerves. Theoretically, therefore, the elements which produce shock in abdominal operations are irritation of the parietal peritoneum and traction on the abdominal viscera. The nearer the viscera are to the diaphragm the greater the degree of this effect. Exposure of the abdominal viscera to air has a bad effect chiefly due to loss of temperature. When viscera are taken out of the abdominal cavity traction and loss of heat act together. These theoretical conclusions based on our knowledge of the sensibility of the abdominal viscera are borne out by Crile's experimental work and clinical observations. Manipulations in the pelvis, or rather of its viscera, produce very much less effect than on the higher organs. In no field of surgery can its art be better employed in lessening the degree of shock than in the abdomen, and it is very satisfactory to find that the conclusions of clinical observers are confirmed by scientific laboratory experiment. In no other field is the method of anæsthesia of greater importance. In weak patients the skin incision can be made with local infiltration; the muscle opening, whatever its nature, can be performed without pain by the proper intraneural method of Oberst, so well illustrated in Harvey Cushing's procedure for inguinal hernia. General anæsthesia is now given for the division of the parietal peritoneum. Throughout the remaining intra-abdominal operation the anæsthesia deepens when any manipulation is done which would cause pain if the patient were awake; for example, traction, separation of adhesions from the parietal peritoneum;

when abdominal sponges are introduced or removed if they come in contact with the parietal peritoneum. During the operation *per se* on the viscera themselves, the narcosis can be very light; for example, throughout the entire suture of a gastro-enterostomy practically no anæsthetic need be given. Again, when the abdominal wound is closed, the narcosis must be made deeper. In long abdominal operations and in operations upon weak patients vigilant attention to the most minute details of narcosis and manipulation of the tissues is the chief factor which insures success. In no other operation is shock a more disastrous complication. It may be fatal of itself. If not, it so lowers the resistance of the patient that post-operative complications are more frequent. The greater one's experience in intra-abdominal surgery the more is one impressed with these facts.

Spleen.—According to Lennander the capsule and the parenchyma of the spleen are insensible. In operations under local anæsthesia this organ can be handled with impunity, providing one does not touch the parietal peritoneum in the neighborhood or make traction upon the spleen. This traction irritates the diaphragm through the ligament, and the posterior peritoneal connective tissue with its nerves through the splenic vessels. Crile in his experimental work on dogs found no change in the blood pressure in operations upon the spleen. Splenectomy, therefore, is of itself an operation in which shock need be feared only if there is hemorrhage or necessary prolonged and vigorous tension.

Pancreas.—Crile records no observations on manipulations of this organ. Lennander finds the parenchyma of the pancreas insensible. In practical surgery, however, injuries and operations in the region of the pancreas are frequently attended with an unusual degree of shock. This undoubtedly is due to the numerous branches of spinal nerves in the peripancreatic tissue. In rupture of the pancreas from contusion or wound and in the so-called pancreatic apoplexy, in which the pancreas and the peripancreatic tissue are infiltrated with blood from the ruptured artery, the patients exhibit a degree of shock far out of proportion to the loss of blood. This general condition can be explained by the irritating effect of the pancreatic juice escaping into the peritoneal cavity, which excites a chemical peritonitis associated with a hemorrhagic exudate. Without much doubt, in addition to the depressing effect of this irritant, there is also a toxic element. The same is true of acute hemorrhagic pancreatitis in which the general symptoms—a combination of shock and intoxication—are far out of proportion to the local infiltration. For example, a similar hemorrhagic and inflammatory exudate about the kidney or the rectum is never associated with such a degree of general depression. For this reason, in operations in the region of the pancreas, injury of this organ must be avoided, and if incision or partial excision of the pancreas is necessary the peritoneal cavity must be protected from the pancreatic juice.

Liver.—The peritoneal covering of the liver and its substance, according to Lennander, are insensible. This I have confirmed in local-anæsthetic operations on the abdomen. An abscess of the liver, after it has been walled off by gauze, can be opened secondarily with knife or Paquelin cautery without pain. Practically, however, in operations in the region of the liver, it is difficult to handle the organ without some traction on the diaphragm. This produces pain if the patient is not under general narcosis, and becomes a factor in producing shock in certain cases. The excision, however, of even large pieces of liver with knife or cautery can be performed without any danger of shock from this manipulation. The only element of danger is hemorrhage.

Kidney.—The intimate capsule and parenchyma of the kidney are insensible. Crile in his experiments found that in cutting, contusing, or crushing the kidney no effect was observed upon the blood pressure, except when during these manipulations parietal peritoneum was injured. Nephrectomy of itself had very little effect upon the blood pressure. In practical surgery, however, shock frequently attends operations upon the kidney. The degree of shock is in a fairly direct proportion to the amount of manipulation necessary to free the kidney. If the diseased kidney, tumor, or inflammation is very adherent to the perinephritic fat and connective tissue or the neighboring parietal peritoneum, the manipulations to free the kidney necessarily involve rough handling of sensitive tissue. Considerable traction is required on the abdominal wound. In these cases, even though there be but slight loss of blood, shock can easily be recognized clinically. Quite frequently in these cases a considerable amount of oozing cannot be prevented. For these reasons many of the patients are quite shocked. It is in this group of operations that rapidity becomes a very important element in preventing shock. When by performing the operation slowly and carefully the surgeon can lessen the sensory impulses which produce shock, time need not be considered. However, when he cannot avoid this rough handling of tissues, as in the enucleation of an adherent diseased kidney, shock is less in direct proportion to the rapidity of the operation. The anuria that may take place as a post-operative complication of kidney surgery apparently cannot be based upon shock as a factor.

Testicles.—The testicle, epididymis, and its intimate peritoneal capsule are insensible according to Lennander. The sensory nerves are present in the skin, dartos, and external coverings of testicle and cord. The older views that castration was associated with an unusual degree of shock have proved to be fallacious. According to Crile an effect upon the blood pressure is only observed when rough manipulations are made upon the external coverings or when one dissects a very adherent hernial or hydrocele sac. Therefore, in operations for hernia, hydrocele, and upon the testicle rough manipulations should be avoided. In old people with strangulated hernia the operation should be always done under cocaine infiltration. In some cases no attempt

should be made to excise the sac, because, on account of its size or adhesions, these manipulations become elements of danger in increasing shock. When the sac is not adherent and can be enucleated without difficulty, there is no increased danger in its removal.

Spinal Column.—Crile demonstrated that in operations of laminectomy no change was observed in the blood pressure until the membranes of the cord were exposed. Contact, however, with sensory nerve roots showed the most marked change. In my limited experience with laminectomy I have been surprised at the absence of much shock. Undoubtedly the skin, muscles, and deeper tissues, if one confines the incision to the middle line, contain very few sensory nerves. Crile demonstrated this in laminectomy under cocaine infiltration. Nor did his patient experience pain when the spinous processes and lamina were divided.

Extremities.—In amputations the chief factors which produce shock are the divisions of the skin and nerves. This can be prevented by cocaine injections. That tearing of the skin and of the nerves are definite factors in producing shock is confirmed by observations in accident surgery. All patients with lacerated, contused, or crushed wounds of the extremities are shocked in direct proportion to the injury of the skin and larger nerve trunks. The shock may be extreme without the loss of blood. There is no evidence to indicate that the crushing or fracture of the bone is of itself a factor. For example, in extensive comminuted fractures without complicating injuries of the soft parts there is no shock. The treatment of patients with extensive injuries of the extremities and shock is one of the important problems of traumatic surgery, and will be discussed later.

Duration of the Operation.—From the preceding discussion one can easily understand that the time of the operation becomes a distinct element in shock only when during this time manipulations are made which produce sensory impulses. As stated before, when discussing kidney operations, it is better to prolong the operation, if by this these sensory impulses can be avoided. When, however, the manipulations necessary for the operative procedure are factors which, we know, will produce shock, the time of the operation should be shortened as much as possible. Irrespective of the manipulations, there are two other factors which produce shock, which must be borne in mind, as they are increased by the duration of the operation: first, the general anæsthetic, second the lowering of the temperature by exposure of large areas of fresh tissues to the air. In long operations the quantity of the anæsthetic can be greatly reduced by the so-called method of interrupted narcosis, which I discussed under operations upon the abdomen. The tissues can be protected by moist warm gauze. Within certain limits of time, I do not believe that the general anæsthetic or the exposure of tissues to air is as important a factor in producing shock as the rough handling of tissue. It frequently, then, be-

comes a choice of evils, and personally I would prefer a little longer operation for a gentle dissection, bloodlessly, to a shorter operation with more hemorrhage and rough handling.

Anæsthesia: The Relation of Anæsthesia to Shock.—Continuous anæsthesia alone will kill animals. Chloroform is a more potent factor than ether. Crile in all of his experimental work on animals considered that the general anæsthetic was always a factor. Blauel (*Beiträge zur klin. Chir.*, 1901, vol. xxx., p. 271) was one of the first to contribute extensive observations on blood pressure during ether and chloroform narcosis. When other factors are eliminated the arterial tension during ether narcosis is well maintained and usually slightly increased, while in chloroform narcosis there were observed great fluctuations and, as a rule, a lower blood pressure. Chloroform, therefore, should never be given in shock or in any cases in which the lowering of the vasomotor tone would be dangerous to the patient. The most recent communication on this subject is by Mueller (*Archiv f. klin. Chir.*, 1905, vol. lxxv., p. 896, and vol. lxxvii., p. 420). Mueller's observations are concerned chiefly with mixed narcosis. He has demonstrated to his own satisfaction that oxygen is a very important, perhaps essential gas to combine with every anæsthetic. In an oxygen-chloroform narcosis the depression and fluctuation of the blood pressure are less marked than with simple chloroform. The oxygen-ether narcosis gives better results as regards blood pressure than the simple ether or oxygen-chloroform. In some cases the best results were obtained with a mixed oxygen-ether-chloroform narcosis given with a special apparatus. I have had no personal experience with these mixed general narcoses, nor with the various apparatus used for their introduction. In my own experience I have so far been satisfied with ether given by the drop method on an ordinary chloroform mask. This has proved the most satisfactory anæsthetic in cases of shock or in weak patients for whom a general narcosis was absolutely necessary. A patient in a condition of shock requires very little anæsthetic. One seldom, if ever, observes the cyanosis and asphyxia which now and then are a complication in robust individuals. I always combine, if possible, a local cocaine infiltration with the general narcosis in cases of shock or in patients in whom I anticipate shock. Further investigation undoubtedly should be made to demonstrate if Mueller's conclusions are correct. If they are, oxygen and its combination with ether, or ether and chloroform, should be employed.

Local Anæsthesia.—There is no doubt that if the operation can be performed painlessly under local anæsthesia there is less shock than if a general narcotic is employed. But, it is very important to remember that the danger of a general anæsthetic is less than the prolonged, *painful* attempt under local anæsthesia. In the early years of local anæsthesia many of its advocates were so enthusiastic that they did not seem to appreciate that some manipu-

lations were painful. Strong individuals can stand it, and there is no question that for these patients the dangers of pain during the operation are less than the dangers of a general anæsthetic. But this is not true in patients suffering from shock, or in weak, young, or very old individuals. In these latter cases one should attempt as much as possible with local anæsthesia, but for painful manipulations general narcosis is indicated. For the proper procedure in these cases one will find the observations of Lennander on the sensitiveness of tissues and organs of the greatest value.

Spinal Anæsthesia.—With this method I have had no personal experience, but since its introduction I have interested myself thoroughly in its literature. There are no observations to indicate that it produces shock. Its dangers are due to the toxic effects of the cocaine introduced subdurally. A patient intoxicated with cocaine undoubtedly has a lower resistance. Blood-pressure records in spinal anæsthesia, if made, have not been published to any extent, until the communication from Bier's clinic by Mori (*Deutsche Zeitschrift f. Chir.*, 1904, vol. lxxiv., p. 173). In these observations the spinal anæsthesia was produced by the new method of Bier in which adrenalin is employed with cocaine. No blood pressure observations were made in those cases which were anæsthetized with cocaine alone. Mori found very little in the literature on the effect of cocaine injected intraneurally. His observations demonstrate that the blood pressure in spinal anæsthesia produced by adrenalin and cocaine is not as well maintained as that observed after a general ether narcosis, but is very much better than that observed after chloroform narcosis. There is no evidence from this observation alone that spinal anæsthesia is a better method than ether narcosis for shock. The experimental work of Schieffer, also from Bier's clinic (*Deutsche Zeitschr. f. Chir.*, 1905, vol. lxxvi., p. 581), however, is very suggestive. He demonstrated that if animals are shot from a distance of from 30 to 40 metres they fall and do not rise; that is, the immediate effect of the contact of the shot is out of proportion to the actual injury. When, however, these dogs are first anæsthetized by spinal anæsthesia the immediate shock is either not present at all or is very much reduced. Klapp believes that spinal anæsthesia blocks the afferent sensory impulses in very much the same manner as they are blocked by an injection of a nerve trunk. It will require further investigation to determine whether these findings can be utilized in practical surgery. It is suggestive, however, that for extensive injuries of the lower extremities it might be a good plan immediately to block further sensory impulses by an intraneural injection and then proceed with the necessary amputation.

Scopolamine-Morphine Anæsthesia.—At the present time I have been unable to find any blood-pressure records made when this method of anæsthesia has been employed, or to learn whether it has any advantage over cerebral or spinal narcoses in shock.

Hemorrhage.—According to Crile loss of blood always predisposes to shock, and when it is considerable, even if it cause but little depression in the blood pressure, the animal does not withstand a rather severe or protracted operation. Hemorrhage from venous trunks caused the most profound impression. In practical surgery hemorrhage is a very important element in shock. In my own experience I never feel the same anxiety when patients exhibit symptoms of shock if there has been no loss of blood. If, however, there has been considerable loss of blood, the appearance of symptoms of shock should be regarded as an indication to cease further operative manipulations at once, if possible. For example, in cases of osteomyelitis in children, situated in the upper portion of the femur, where an Esmarch cannot be used, the operation in some cases has had to be performed in two or more sittings. The bleeding from the involucrum is always considerable and, without an Esmarch, cannot be checked during the necessary chiselling to explore the infected medullary cavity. I have records of at least five cases in which the condition of the patient was sufficiently threatening to indicate immediate packing of the wound and a postponement of further operation for some days. In these cases the operation has been completed in two or more sittings, with recovery of the patient.

Clinical Observations on the Various Factors which Produce Shock.—In the previous discussion on the experimental work it was impossible not to discuss from time to time the confirming clinical observations. Unfortunately, at the present time I cannot obtain a sufficient number of blood-pressure observations taken before, during, and after various operations to compile conclusions of practical value. At the present time, few, if any, surgeons have had sufficient experience with blood-pressure records to depend upon them for an estimation of the patient's condition during the operation. In the majority of instances a surgeon of experience can judge pretty accurately the general condition of the patient, and, as a rule, seldom loses a patient from shock. This estimation is not based upon a single factor. His careful study of the case before operation gives him an estimate of the strength of the patient; his knowledge of the sensibility of the tissues to be manipulated at the necessary operation gives him a fair idea of the amount of shock he will produce. As the operation proceeds, the character of the respiration and pulse and the color of the skin and lips indicate how the patient is standing the operation. It is not so much the rapidity of the pulse or respiration, as the comparative frequency of the pulse and respiration, during the operation, that indicates how the patient is withstanding the ordeal.

In the majority of cases subjected to operation the general condition of the patient is such that, if the anæsthesia is properly given and the operation carefully performed without the loss of blood, the danger of shock is so insignificant that it need not be considered. In these cases we do not need an

instrument of precision to record the blood-pressure, although records in these cases should be made for their value in a comparative study.

In a smaller group of cases the significance of shock is of the greatest importance. Various factors come into consideration.

Anæmia.—Patients with secondary anæmia, especially if the hæmoglobin is low, are not good subjects for operation. All the factors which produce shock apparently act in these cases with greater severity—the general anæsthetic, slight loss of blood, all operative manipulations are never borne as well as in individuals with a normal blood count. This subject I have discussed with the literature in *Progressive Medicine* for December, 1901, p. 207.

It is, therefore, very important for the surgeon to insist upon a complete blood count in all those cases in which there is clinical evidence of anæmia. Theoretically a blood count should be made in every instance. I believe it is more important than an examination of the urine. In practice it is not done.

Diabetes.—There is considerable literature on the results of operation in patients suffering with diabetes. I am inclined to think that the dangers are somewhat overestimated. Undoubtedly the diabetic patient has a lowered resistance, and it is sometimes difficult to estimate the advent of diabetic coma. Undoubtedly, in cases suffering with diabetes, one would undertake an operation only when absolutely necessary. In my own experience three cases of appendicular abscess suffering with diabetes took the anæsthetic well and exhibited no symptoms of shock after the short operation necessary to drain the abscess. In a number of patients with gangrene of the extremities due to arterio-sclerosis in which there was also glycosuria I observed that the anæsthetic was well taken and there was no extreme degree of shock after the necessary amputation. One, however, approaches an operation on a diabetic patient with great caution and uses every means to lessen the quantity of the anæsthetic administered and shorten the operation.

Nephritis.—Operations are seldom performed when the clinical picture of this disease is established. In looking over the records I find a number of cases in which albumin and casts were present in the urine without any other definite symptoms of nephritis. As a rule, local anæsthesia has been employed in such cases whenever possible. In studying the histories of these cases and a few with definite nephritis, as well as the literature on decapsulation of the kidney for different forms of acute and chronic Bright's disease, one is impressed with the fact that these patients take the anæsthetic well and are not more shocked than patients without these kidney lesions. The danger apparently is not from shock, but from the effect of the anæsthetic and operative manipulations on kidney function, a subject to be discussed elsewhere in this system.

Therefore, in this group of cases the same care should be employed as that used in patients suffering from shock or in whom we fear shock, because these preventive measures are important to lessen both dangers.

Alcoholism.—My experience in surgery on this class of patients is limited, nor am I familiar with this literature. One is chiefly impressed that this class take the general anæsthetic badly. The dangers of general narcosis are always greater, and therefore, as narcosis is always a factor in shock, it is exaggerated in patients addicted to the excessive use of alcohol. There are sufficient blood-pressure records to demonstrate that alcohol is of no value as a stimulant in shock. In large doses it is a depressant, and for this reason, in accident surgery, if the patient comes to the surgical clinic, as they frequently do, overdosed with whiskey given them as a "first-aid" measure by the ignorant, this intoxicated state must be borne in mind and considered in the treatment. As far as my own experience goes, acute and chronic alcoholism must be regarded pathologic conditions in which the patient is less resistant to all the factors which produce shock. Alcohol is contraindicated in the treatment of shock.

General Infection.—It has been my personal experience that patients suffering with general infection react more quickly to all the factors that produce shock, and this must be borne in mind in all operations. A high temperature and toxins, whatever their character, apparently after a time affect the centres in very much the same manner as do the sensory impulses which produce shock. Whether these are of an inhibitory or of a depressant character has not been demonstrated. Every one is familiar with the rapidity with which shock manifests itself when patients are operated upon for general peritonitis, and how much more careful one must be in performing the necessary abdominal manipulations. In these cases the patient may suddenly become almost pulseless when the intestines are removed from the peritoneal cavity. On the other hand, in the normal individual one may keep the intestines out of the abdominal cavity under tension for a relatively long time before symptoms of shock manifest themselves. Amputations for infected compound fractures or for any infection of the extremities are associated with a relatively greater degree of shock than amputations for tumors. It is important, therefore, to bear in mind that operations upon patients suffering with general infection must be conducted on the supposition that shock is a very dangerous factor.

Local Infections.—According to Lennander, in all tissues and organs supplied by sensory nerves from the cerebro-spinal system the sensibility is increased by an inflammatory lesion. Practically, this knowledge is not of very great importance in relation to shock. It is better, however, to handle these inflamed sensitive tissues more gently. On the other hand, all tissues and organs not supplied by these sensory nerves, but by the sympathicus and lower vagus, are no more sensitive when they are the seat of disease, and for this reason they can be handled with just as much impunity.

In local anæsthesia it has been demonstrated that inflammation of sensitive tissues increases their sensitiveness to such a degree that, in the majority of

instances, unless the nerve can be blocked above the area of inflammation, an operation cannot be performed under this method of anæsthesia.

Starvation.—I use this term to define a condition of loss of weight and strength attributable to defective nutrition, no matter what its cause. We observe the extreme degrees more especially in strictures of the œsophagus, carcinoma of the stomach, pyloric stenosis, and chronic obstruction of the small and large intestines. Undoubtedly, in the cases of pyloric stenosis and chronic obstruction of the intestine lower down there is another factor, that of auto-intoxication. These patients are all bad subjects for operation. They quickly react with shock to every factor—anæsthesia, duration of operation, hemorrhage, manipulation of sensitive tissues and organs. In this group, perhaps more than any other, the surgeon must use all the means at his command to prevent a fatal condition of shock.

Auto-intoxication.—This term is used to define a condition of general infection due to the absorption of toxins from the alimentary tract. Its acute form is observed in all cases of acute intestinal obstruction; the chronic form in all cases of pyloric stenosis and chronic intestinal obstruction. Patients suffering from this toxæmia are bad subjects for operative intervention.

Jaundice.—The chief danger of surgical intervention on patients suffering with obstructive jaundice is that of secondary hemorrhage. In looking over the records of operative intervention upon patients suffering with jaundice due to stone in the common duct I have been unable to find any positive evidence that they are bad subjects for anæsthesia and the necessary operative manipulation because of the jaundice alone. Those cases in which there are, in addition, marked anæmia and loss of weight, exhibit greater reaction to the operation than those cases in which the jaundice is even more intense, but in which there is no anæmia or loss of weight. It is the duration of the jaundice, and not its intensity, that chiefly affects the general condition of the patient and lowers his resistance. The operative manipulations necessary to expose the common duct, especially if there are adhesions, are of a character that undoubtedly, if prolonged, or performed upon patients with lowered resistance, produce shock. This should be borne in mind.

Acute Hemorrhagic Pancreatitis.—In the clinical picture of the early hours of this disease shock is a prominent feature. In the discussion of the operative treatment of this lesion there has been much difference of opinion as to whether intervention is justifiable on account of this condition of depression verging on collapse. In these cases, if an operation is decided upon, every effort should be made to shorten the anæsthetic time and to limit the abdominal manipulations.

Shock Associated with Injury.—In accident surgery many patients are admitted to the clinic in a condition of shock the degree of which varies. When an operation is demanded on account of the nature of the injury, the surgeon must not only recognize that shock is present, but must estimate its degree.

Shock and Hemorrhage.—In accident surgery the first important factor to be estimated is whether the shock is due to hemorrhage or is simply the result of injury. This differential diagnosis is of chief importance when the injury is subcutaneous, in the chest or abdomen, or about the great blood-vessels in the axilla, groin, or limbs. Hemorrhage must be checked by immediate operation, no matter what the degree of shock. This differential diagnosis, when the possible area of hemorrhage is concealed, is frequently difficult. When the injury is in the region of the axilla, groin, or the limbs the presence of a rapidly increasing swelling is a definite indication of vessel injury and the formation of a hæmatoma. A decision in regard to operative intervention in such cases is frequently very difficult. Experience has demonstrated that in a certain number of cases the tension of the surrounding tissues limits after a time the size of the hæmatoma, that thrombosis takes place in the ruptured vessel, and accomplishes a stoppage of the hemorrhage which for the time has threatened life. In these cases the operative manipulation is usually of sufficient magnitude to contraindicate its immediate performance. For this reason it should never be performed unless the indications are that thrombosis is not taking place in time to save the patient from death from loss of blood. The degree of shock in these cases is the best sign of an indication for operation.

The differential diagnosis between shock from abdominal contusion without hemorrhage, and that with hemorrhage from rupture of the viscera, is a very difficult one. In the former, operation is contraindicated; in the latter, immediate laparotomy is imperative.

Experience has demonstrated that ruptures of the liver and spleen and large vessels of the abdomen have not the same tendency to spontaneous cessation by thrombosis. Here the blood escapes into the free peritoneal cavity. The conditions favorable to thrombosis which are present in the rupture of the large vessels of the extremities are absent in the abdomen.

As a rule, an injury of sufficient force to produce a rupture of the abdominal viscera would of itself cause shock. In my own experience the degree of shock is never as great as in those cases in which there is, in addition, hemorrhage. For this reason an accumulated experience enables one to make the proper diagnosis in the majority of cases. Movable dulness in the flanks is pathognomonic of hemorrhage; it is not always present. A blood count theoretically should be of value, but experience has demonstrated that, as a rule, the blood-changes do not take place quickly enough after the hemorrhage to be of aid in the necessary immediate diagnosis. The leucocytosis of hemorrhage does not appear until some hours after the accident. The aid of a blood-count unfortunately cannot be depended upon in these cases. In the literature the recoveries after immediate laparotomy for rupture of the spleen, liver, and pancreas have been due to an immediate intervention based upon the history of the injury and the clinical picture of shock. Now and then an abdomen is

opened unnecessarily. But if the exploratory exposure of the abdominal cavity is performed under local anæsthesia, there should be no mortality in the cases with negative findings. Unfortunately, I am unable to present any blood-pressure studies in these cases to demonstrate that measurements are of diagnostic value. I am inclined to think that they would be.

In rupture of the kidney the problem is perhaps a more difficult one, because experience has demonstrated that in quite a number of cases the hemorrhage ceases spontaneously. Yet, in other cases, life has apparently been saved by immediate incision and packing, or, in a few cases, by nephrectomy with ligation of the renal vessels. In this group the indications for operation are based upon the degree of shock, the size and increasing development of the perirenal hæmatoma.

In contusions of the chest with hemorrhage from the intercostal vessels or ruptured lungs the problem of operative intervention is a very difficult one to solve. My own experience has been limited, and the literature is somewhat scanty. The same principles apply as those discussed under rupture of the kidney, or a subcutaneous injury of a large blood-vessel of a limb. In the intrathoracic hemorrhage a certain number of patients recover without operative intervention. In others the hemorrhage must be checked by exposure of the bleeding vessels. Here again the degree of shock is of aid, and the increasing area of thoracic dulness indicates the amount of hemorrhage. The results of intervention in cases of rupture of the intercostal vessels have been good; on the other hand, the results obtained in cases of rupture of the lung are not very encouraging.

All of these cases should be watched very carefully, and when the condition of shock gradually grows worse and the physical signs indicate an increase of the hæmatoma there should be no further delay. This group of cases demonstrates how important it is for surgeons to study very critically shock in all of its clinical manifestations, and how much we are in need of blood-pressure and blood-count records.

Amputation During Shock.—This subject has been one of discussion from the beginning of surgery. In all injuries of the limbs associated with an extreme degree of shock, and in which the nature of the injury demands either amputation or some other operative manipulation, the question at once arises whether the best results are obtained by immediate action or whether the patient's chances of recovery are better if the operation is delayed. Experience has demonstrated that there is no fixed rule. In a certain number immediate operation must be performed, in others it is better to delay. The duration of the time of postponement varies. Hemorrhage is always an indication for immediate intervention and must be checked, even though it demand an extensive operation. The danger of further loss of blood is much greater than the danger of an operation in a condition of shock. If hemorrhage has

ceased, operation may be indicated, because the pain from the mutilated limb is sufficient to be considered as a factor which will increase shock. When these two factors are absent, it is better to delay in order that the patient may have the opportunity to recover as much as possible. The operation, however, must not be postponed too long, because after a time the element of infection becomes an important factor.

Wainwright, of Scranton, Pennsylvania, has had a large experience in traumatic surgery and has contributed to the question under discussion ("Clinical Studies in Blood-Pressure and Shock in Traumatic Surgery," *Medical News*, New York, March 25, 1905).

He writes: "To remove the nerve impulses after trauma, an immediate repair of the injury, if at all feasible, is very important. For this reason our own view is strongly in favor of primary amputations in limbs hopelessly mangled. Leaving a mangled, oozing limb with crushed and exposed nerves, in the hope that delay will give a more favorable opportunity for intervention, will, in many cases, by allowing the cause continually to act, only drive the patient into a condition beyond all hope. A well-covered stump with oozing checked, on the other hand, will give a chance to a patient in whom the cause of shock is stopped and to whom the administration of therapeutic measures will not be like pouring water through a sieve."

There are cases, however, in which there have been a great deal of injury and much loss of blood, and in which the degree of shock is severe, that apparently do better if the operation is delayed, providing the two indications just discussed—hemorrhage and pain—are not present.

It has been my observation that in traumatic surgery the patients exhibiting a definite clinical picture of shock do, as a rule, better than those in whom the clinical picture of shock is less evident. The former receive appropriate treatment, the latter are apt to be treated on the supposition that shock is absent. In looking over the records of the Johns Hopkins Hospital Surgical Clinic of cases of primary amputation for compound fracture and other crushed injuries of the extremities, I find a few examples of death a few hours after the amputation, from shock. In examining the notes of these cases we are impressed by the fact that, if shock was present before the operation, it was not recognized. They received no preliminary treatment, the operations were not hastened, but a careful, painstaking amputation was performed.

On the other hand, the patients in whom the local injury was more severe and the clinical evidence of shock was unmistakable have recovered. These patients received preliminary treatment, and the amputation was rapidly performed—in the older cases under primary, short anæsthesia; in some recent cases, under anæsthesia associated with blocking of the nerve trunks. No attempt was made to do anything more than rapidly to remove the mutilated limb, cutting through uninjured tissue.

The practical deduction from this is that in all cases of traumatic surgery the possibility of shock must be borne in mind. The clinical picture is often obscure; we have no means of estimating accurately the exact quantity of blood lost before admission to the clinic. For this reason it is better to treat these patients on the principle that shock is present to a considerable degree and that a great deal of blood has been lost before the patient came under observation.

In my own experience loss of blood is the most dangerous factor in these cases of traumatic shock.

Exposure to Cold.—Contributions from military surgeons indicate that exposure to cold is a distinct element in increasing the degree of shock. This factor is frequently present in traumatic surgery.

Exposure to Heat.—That burns may produce an extreme degree of shock has been discussed. I am unable to find any literature giving observations on the relation of shock to high temperatures; that is, whether injured persons in very hot climates exhibit a degree of shock out of proportion to the character of the injury and the loss of blood. In a considerable experience of my own in operations during the severe heat of the summer in Baltimore I have been unable to find that there is any increased mortality, but on days on which the temperature has been very high—over 90 or 95 degrees—I have been impressed that a certain number of cases show more evidence of shock at the end of a long operation, and I have quite frequently postponed operations of unusual magnitude on account of the great heat. During this extreme heat it has been my rule to have ice caps placed on the head of the patient during the operation. I have observed a few cases of heat stroke during operation and a few of heat collapse. I have never been called upon to operate upon a patient suffering with heat prostration or heat stroke. In alcoholics I am quite convinced that delirium tremens is much more frequent during the hot season of the year, after operations, especially for injuries. When secondary operations are necessary, for example, for infected compound fracture, these patients are unusually bad subjects. I believe that in critically ill patients extreme degrees of heat in the operating-room should be considered a factor which may increase the shock, and precautions should be taken for protection—ice caps to the head, less covering to the body. If possible, the operation should be postponed, or performed at night when it is cooler.

Atmospheric Pressure.—Theoretically the blood pressure should be affected to a certain degree by the atmospheric pressure. Whether this is ever a factor in shock in critically ill patients I am not prepared to say, nor have I been able to find any investigations on this point.

Psychic Factors.—To what extent mental conditions can produce shock, or exaggerate it when present, is very difficult to determine. One frequently observes syncope in strong individuals from the loss of blood. Fear

may produce a general condition bordering on shock. This question has not been investigated, from a scientific standpoint, in its relation to practical surgery. In my own experience all these mental conditions, which may be classified under the term fright, anxiety, nervousness, exaggerate the clinical picture of shock when it is present. But apparently they are not factors of such importance as those already discussed. When these patients are narcotized the pulse and respiration immediately improve. The importance of studying this question is chiefly from the standpoint of diagnosis. The surgeon might easily attribute the general condition of the patient to factors other than mental, and thus be led to erroneous deductions as to the presence of shock, or as to its degree. This mental factor must always be borne in mind, but it is a dangerous mistake to attribute to fear or nervousness the general condition of the patient when in reality it is due to more serious conditions. This mistake is much more to be guarded against than the reverse.

I have never been able to convince myself that these mental factors are ever of sufficient significance to influence the results, as far as mortality is concerned, of traumatic or any other form of surgery. Nevertheless, every effort should be made on the part of those in attendance upon the patient to allay fear, and calm all nervous or other anxieties. The ability to restore the patient to a quiet and confident frame of mind may not improve the immediate and permanent results of the surgical intervention; nevertheless, it adds so much to the comfort of the patient that every effort in this direction should be made.

In operations under local anæsthesia we observe the good effect of this attitude toward the patient. It has been called "moral anæsthesia." The surgeon who learns the art of this method can perform under local anæsthesia many operations for which others have had to employ general anæsthetics. This is well illustrated in operations for exophthalmic goitre. If the surgeon gains the confidence of his patient and is able to control the nervous element during the operation, everything goes smoothly. When, however, this control is lost, the general condition of the patient immediately gets worse—he becomes restless, the pulse increases in rapidity, the respiration is labored, and not infrequently it is impossible to finish the operation without a general anæsthetic. By reason of its relation to shock I believe it is safer to use general anæsthesia if during the attempt under local anæsthesia the surgeon is unable to calm and control his patient by the so-called "moral anæsthesia."

Younger surgeons especially are apt to underestimate these mental factors. Their attitude toward patients is frequently one that contributes not at all to the mental comfort of the latter.

Tact and cheerfulness compatible with the seriousness of the patient's condition are the two most important attitudes on the part of the surgeon. At the same time he should never allow himself to exhibit any anxiety or give utterance to any expression which could be interpreted as uncertainty in regard

to the treatment or its result. As stated before, we have no definite evidence that this so-called moral anæsthesia or treatment materially affects the ultimate result.

It does, however, affect the comfort of the patient, and, I am quite convinced, is one of the most important factors in preventing or lessening a post-operative complication which, for the lack of a better term, has been called "post-operative neurosis."

Summary of the Etiological Factors in Shock.—The most important are sensory impulses affecting the medullary centres, the next is hemorrhage. General anæsthesia, the duration of the operation, extreme degrees of heat and cold, certain drugs, must be considered additional factors. Although their influence has not been proved, psychical effects should be borne in mind. As general conditions which predispose to shock, or associated with which the important factors of shock act with greater effect, we must bear in mind anæmia, diabetes, nephritis, alcoholism, general infection, local infections, all those conditions which interfere with metabolism and nutrition, collected under the term "starvation," and auto-intoxication.

The sensory impulses which produce shock may be the result of traumatism, or the result of cutting, tearing, or mutilating tissues during an operation.

Only those organs and tissues which are supplied by sensory nerves of the cerebro-spinal system need be considered in relation to shock. Their sensibility is increased by inflammatory lesions.

Organs and tissues innervated by the sympathetic nerves or the lower vagus are insensible, and at the present time we have no evidence that their manipulation or injury need be considered as factors in shock. Tumors, according to Lennander, are insensible. In handling these insensible tissues it is important to be familiar with their anatomical relation to or connection with surrounding sensitive tissues.

Drugs and manipulations which in experimental investigations and clinical observations produce a rise in the blood pressure may and do affect the vasomotor centres in a deleterious sense when these centres are exhausted. Overstimulation of these centres may be just as dangerous a factor as a primary depressant or inhibitory action. This is important to recollect in interpreting the readings of a blood-pressure chart. The best index to the good condition of a patient during an operation is uniform pressure. Fluctuations in the curve should be considered indications of exhaustion. Manipulations which produce a sudden and considerable rise in the blood pressure should be interpreted as overstimulation—factors which have a tendency to produce shock. It is quite true that drugs like chloroform, or any manipulations, or loss of blood, which produce a primary fall in the blood pressure, are more dangerous factors than those which produce a primary rise, but both must be considered factors in producing shock. In the employment of the tonometer, or other blood-

pressure-measuring contrivances, the best indication of the good condition of the patient is a *uniform pressure.*

DIAGNOSIS OF SHOCK.

One may classify surgical patients suffering from shock into three groups: those in whom shock is associated with traumatic injuries, those in whom it is associated with some disease, and, finally, those in whom the shock is dependent upon operative intervention. The clinical pictures of shock in all three groups are very much alike. However, the knowledge of the previous history of the patient is of great value in determining the probability of shock and estimating the psychical element and differentiating it from the physical.

Extreme degrees of shock are not at all difficult to appreciate. The moderate degrees and the conditions which predispose to shock are frequently very difficult to recognize; and yet a diagnosis in this stage is of the utmost importance.

Crile differentiates between shock and collapse. He writes that the term *shock* should be used for that condition in which the essential phenomenon is a diminution of the blood pressure and the etiology of which is an exhaustion of this centre of varying degrees due to too frequent and too powerful afferent stimuli. The term *collapse* should be confined to those cases in which the essential phenomenon is a sudden fall of blood pressure due to hemorrhage, injuries of the vasomotor centre, or cardiac failure. In shock, therefore, we have an exhaustion of the centre; in collapse, a suspension of function. Practically, it is very difficult to differentiate an extreme degree of shock from collapse.

In shock in traumatic surgery the knowledge of the amount of blood lost and the nature of the injury is of great value. In shock associated with disease a correct diagnosis of the lesion and an accumulated experience with operations upon individuals suffering with a similar disease are the most important aids in estimating the probabilities and degree of shock.

In operative interventions the experimental work of Crile on the relation of blood pressure to the different manipulations upon the different tissues and organs, the observations of Lennander upon the sensibility of tissues, and one's experience on the relation of the different operative manipulations to the general condition of the patient, allow the surgeon to estimate, during the operation, with a considerable degree of accuracy, the condition of the patient, how much more the patient can stand without producing a degree of shock dangerous to life.

When patients are under a general narcosis it is less difficult to estimate the degree of shock. In shock associated with injury and disease—for example, acute pancreatitis, intestinal obstruction, intestinal perforation, general peritonitis, etc., etc.—it is frequently difficult to determine how much is mental and how much physical. It has been my personal experience that the greater the degree of shock the fewer the symptoms which may be classified as mental.

A patient in a condition of shock is quiet, he appears somewhat dazed; although there is no delirium, the action of the mind is slow; there is no nervousness and there are no manifestations which might be called hysterical. The pulse, as a rule, is rapid; the blood pressure, if measured, will be found low. The skin and mucous membranes are pale. The temperature is frequently below normal. All cutaneous and deep reflexes are diminished, they may be absent. The skin feels cold and as a rule clammy. The respirations are shallow.

A rapid pulse is by no means an indication of shock. It must be interpreted in its relation to other factors. A blood-pressure observation is of the greatest importance to interpret the significance of a rapid pulse. In exophthalmic goitre the pulse is rapid, but the pressure high; and in operations upon patients of this kind one should always employ a tonometer. In nervous and hysterical patients the pulse is rapid, but the blood pressure in the few records at my disposal is normal or slightly elevated.

Cyanosis should be considered a definite indication of the bad condition of the patient.

When the symptoms are chiefly psychical the patient is flushed, restless, anxious, the reflexes are increased, all symptoms are exaggerated. The true interpretation of these symptoms is, as a rule, not difficult. They disappear the moment the patient is under narcosis.

From this discussion it is to be seen that the diagnosis of shock is at the present time not scientific. We have not sufficient observations on blood pressure in practical surgery to allow one to estimate the degree of shock by the blood pressure alone. The diagnosis of shock is an art difficult to describe. It is based upon experience and the proper estimation of various factors.

Practically, if the surgeon will bear in mind all the points previously discussed, he will be able to estimate the condition of his patient before and during operation with sufficient accuracy for the purposes of safety.

PROGNOSIS.

As the result of injury without hemorrhage, death from shock seldom takes place. If a fatal result is at all to follow an injury without loss of blood, death is almost instantaneous. Recovery from shock due to the primary effect of the injury is usually permanent and immediate. Sudden death after an injury should, perhaps, be attributed to collapse, as described by Crile. Sudden death from blows upon the lower chest and epigastrium have been observed in so-called solar-plexus blows, well known in pugilistic encounters. According to Crile's experimental research the collapse is due to the effect upon the heart; the solar plexus may be disregarded as a factor.

Even in the extreme degrees of shock from injury, if the patient shows any symptoms of reaction, the prognosis for recovery is good, provided no further operative intervention is necessary.

The prognosis of shock due to operative intervention depends very much upon the condition of the patient. Recovery from an extreme degree of shock usually takes place if the patient was in good condition before the operation and provided that when the symptoms of shock appear the administration of an anæsthetic and operative intervention can immediately be suspended.

When hemorrhage is one of the factors in shock, whether it be due to injury or to an operation, the prognosis is not so good. Death may not be immediate, but a patient exsanguinated reacts much less quickly, and the dangers of secondary complications are very much greater than they are in those cases of shock which are not associated with hemorrhage.

TREATMENT OF SHOCK.

At the present time the consensus of opinion favors a treatment which is simple and, on the whole, passive. In the presence of shock nothing should be done which, with our present knowledge, may increase the condition. The patient should be kept absolutely quiet, flat on the back, in an elevated position, with the head low. The body temperature should be maintained by artificial heat. Only one drug is indicated hypodermatically—morphine. This is indicated in small doses in all cases. Its quieting effect undoubtedly is beneficial. When the patient is suffering pain, sufficient morphia should be given to relieve this pain. Salt solution given subcutaneously and by enema is indicated in all cases. When there has been hemorrhage the quantity administered subcutaneously should be greater. If the patient's condition is critical the salt solution should be given intravenously; the quantity should vary from 500 to 1,000 c.c. In very critical cases associated with much loss of blood the intravenous infusion should be given rapidly; in patients less critically ill, more slowly. When the shock is not associated with hemorrhage, according to Crile's experimental work, the administration of the salt solution subcutaneously and intravenously has not given evidence of great value. In practical surgery the clinical evidence favors the employment of salt solution in all cases. But, as a matter of fact, in traumatic and operative surgery the majority of cases of shock are associated with hemorrhage, and for this reason salt solution, of course, gives evidence of its great value. In the other cases of shock without hemorrhage, the prognosis is, as a rule, so good that it is difficult to estimate the value of salt infusion. We know, however, both from experimental and from clinical evidence that it is not harmful. For this reason, salt solution should be employed in all cases, intravenously, subcutaneously, or by enemata, according to the condition of the patient.

Crile's experimental work has demonstrated that, in shock, what is required is not a cardiac or a vasomotor stimulant, but some agent which will produce contraction of the peripheral vessels. The chief danger in shock is a dilatation

of the vessels to such a degree that the patient practically bleeds to death within his own vascular system. This contraction of the peripheral circulation can be accomplished by bandaging the limbs and abdomen, or by the employment of Crile's pneumatic rubber suit which accomplishes the same object by increasing the atmospheric pressure. (Figs. 140 and 141).

From the experimental work of Crile and others, adrenalin is the only drug which produces vaso-constriction by its action on the peripheral vessels. Unfortunately, at the present time the clinical evidence in favor of its employment is lacking.

If we were quite certain that this use of adrenalin had no elements of danger, it should be employed in all cases. But at the present time the evidence in favor of its value is not sufficiently positive to warrant us in assuming the risks of its employment, except in desperate cases. Then it should be given intravenously in salt solution and slowly.

In the treatment of patients suffering from traumatic or operative shock I employ position, artificial heat, bandaging of the limbs and abdomen, morphine, and salt solution. I agree with Crile and others that strychnine and cardiac stimulants are of no value and may be injurious.

A certain number of patients with traumatic and operative shock die in spite of all measures for their relief. In these cases there has usually been hemorrhage.

Now and then, during the operation, and less frequently shortly after operation, the patient's condition may suddenly become critical, frequently without previous warning, and death may take place in spite of treatment. These may be considered examples of "emergency shock." The sudden change in the pulse and respiration is so rapid and the evidence of impending death is so manifest that it is difficult not only to ascertain the cause of the collapse, but also to know what is to be done for its relief. In some of these cases it may be the anæsthetic; in others it may be the cardiac shock described by Howell. Crile is of the opinion that in some of the cases the condition is due to a sudden dilatation of the heart. At the present time it is difficult to state whether anything can be done for the relief of these patients. Fortunately, the number of such cases is small. The problem needs further investigation.

Salt Solution.—The solution used for subcutaneous and intravenous infusion in the surgical clinic of the Johns Hopkins Hospital is as follows:

Sodium chloride ($NaCl$)	0.9
Calcium chloride ($CaCl$)	0.01
Potassium chloride (KCl)	0.03
Distilled water (H_2O)	99.06

This stock solution is prepared by the druggist. The nurse in charge of the operating-room takes 50 c.c. of this stock solution and adds it to 950 c.c. of distilled water. This solution just fills a liter glass flask. The flask is corked

with cotton, covered with muslin, which is properly tied to the neck of the flask. These flasks are sterilized by steam under pressure and are ready for use at any time. I do not think that an exact temperature of the solution is necessary. The flask filled with its solution is immersed in boiling water until it reaches a temperature of 100° to 105° F. It is then poured into a glass infusion apparatus or an ordinary rubber douche bag, each provided with a long piece of rubber tubing. Both should be sterilized by boiling. They can be wrapped in towels and sterilized by steam, and in this manner are ready for emergencies. For subcutaneous infusion an ordinary aspirating needle (Fig. 138) is attached to

Fig. 138.

a small piece of rubber tubing in the end of which is a short glass tube. This is boiled with the instruments. When a subcutaneous infusion is indicated the skin along the pectoral border of the breast is cleansed, the flask is filled with salt solution, the needle is attached by telescoping its glass tube end into the long rubber tube, the salt solution is allowed to run out of the needle, the temperature tested on the skin of the arm, the needle is then introduced just below the border of the pectoral muscle, parallel with it in the direction of the axilla. This allows the solution to infiltrate the tissues in the base of the axilla which are very vascular, and absorption takes place rapidly. In ordinary cases 500 c.c. should be allowed to take at least twenty minutes to pass from the flask into the tissues.

The same method is suitable for intravenous infusion, except that a different needle should be employed, one like that shown in the accompanying cut (Fig. 139). As to the locality where the injection should be made, I prefer one of the superficial veins at the bend of the elbow.

Adrenalin Solution.—Brewer of New York advises 15 minims of the 1 : 1,000 commercial solution to 1,000 c.c. of normal salt solution; as a rule, not more than 500 c.c. should be given intravenously; if possible, a blood-pressure

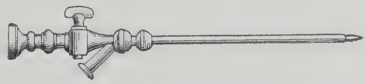


Fig. 139.

apparatus should be employed at the same time. As the blood pressure rises the infusion should be checked. If the blood pressure falls again, the infusion should be resumed. Precordial pain is a contraindication for the further employment of this method.

Wainwright employs the adrenalin solution in the proportion of 1 dram of the 1 : 1,000 solution to 2,000 c.c. of salt solution.

FIG. 140.—Crile's Pneumatic Suit Adjusted for an Operation upon the Neck. View taken from one side.

FIG. 141.—Front View of Crile's Pneumatic Suit Adjusted.

Crile's Pneumatic Suit.— Figs. 140 and 141 illustrate a patient dressed in this suit. It has been placed on the market by the Goodrich Rubber Company of Akron, Ohio, with directions for its employment.

In the treatment of traumatic shock the most important question to decide, if operation is indicated, is, when should this be done? This has been discussed.

In the treatment of shock during operation the most important factors are: the exact knowledge of the patient's condition before operation; a close watching of his condition during operation, so that the surgeon may at once become cognizant of the first symptoms of shock. When these symptoms arise, it is the art of surgery to be able to estimate how much more the patient can stand, because the most important features of treatment are to withdraw the anæsthesia and cease operative manipulations. The routine treatment of shock has not much value, if anæsthesia and operative manipulations must be continued. If continuation of the operation is absolutely necessary, the patient's head should be lowered and an intravenous or subcutaneous infusion given. But a surgeon runs great risks if he continues to give anæsthesia and proceeds with the operation after symptoms of shock manifest themselves.

Any stimulating treatment in the beginning of an operation is contraindicated. It undoubtedly masks and retards the symptoms of shock, so that when the patient does give evidence of shock the condition becomes rapidly more critical.

PART III.

GENERAL SURGICAL DIAGNOSIS.

GENERAL SURGICAL DIAGNOSIS.

By JOSEPH D. BRYANT, M.D., New York City.

The principles in surgical diagnosis are certain fixed, essential truths relating to the diagnosis of surgical afflictions, which truths are the legitimate outcome of surgical experience and experiment, and which are employed by the surgeon to determine the presence and measure the comparative significance of surgical disorders. The principles in surgical diagnosis are properly divided into the general and the special principles. The general principles in surgical diagnosis relate to certain diagnostic truths, to which there are no exceptions within the scope of their application; *i.e.*, pain is a general symptom of surgical affliction. The special principles in surgical diagnosis relate to diagnostic truths having a special relation to certain general or local surgical afflictions, but not necessarily having a like connection with other surgical ills; *i.e.*, pain characterizes neuralgia, and not paralysis.

The constant advance of the science of surgery develops new principles in diagnosis, and also correspondingly lessens the value of principles of former importance, often indeed rendering them inoperative. The general morbid conditions of the human body, whether of a surgical or of a medical nature, have an expression of their own, called the signs and symptoms. Also each special affliction of either condition has its own distinctive form of expression, by means of which it can be recognized from another of the same class. A variety of affliction of a definite sort, with a form of expression common to itself, may be obscured and its presence lost sight of because of the unexpected intrusion of a dissimilar affliction with manifestations peculiar only to itself, called, if you will, a complication of the primary trouble. Also many of the tissues of the human body have each an expression of affliction peculiar to itself; *i.e.*, the serous tissues when inflamed develop a sharp, darting pain, the cutaneous a dull, throbbing pain, etc. Briefly stated, medical and surgical afflictions have each a distinctive language which, when properly interpreted, establishes the diagnosis and indicates the treatment, and, too, often the prognosis, and possibly the sequels of the affliction.

It is believed that the reader will have noticed that surgery and medicine may be so closely associated with each other by common forms of expression as to be quite inseparable; therefore, a surgeon ought to be in most instances as good an interpreter of symptoms as a demonstrator of surgical technique. The surgeon should be fully equipped with practical knowledge, supplemented

by a well-grounded understanding of anatomy, physiology, chemistry, pathology, etc., and a correct estimation of the phenomena relating to the fluids of the body in health and in disease.

Mechanical and other practical devices are as much a part of the outfit of a well-equipped surgeon as of the physician. In fact, no means fitted to aid in determining the essential facts in surgical diagnosis should be absent from the surgeon's armamentarium.

It is not amiss at this time to observe that the powers of human reasoning in diagnostic attainment are apt to be developed in direct proportion to the paucity of other means of reaching final conclusions in the field of differential endeavor. And, conversely, inductive diagnostic attainment should be carefully fostered or it will be disabled by the vigorous assaults made on reasoning effort by the use of the novel expedients employed in diagnosis; not necessarily because of the abundance of these expedients, nor of their presence, but because it is thoroughly human to accomplish a perplexing purpose with as little effort as possible. The scant danger that now attends "explorative incision" is not unlikely in some instances to encourage a degree of mental contentment, inhibiting the reasoning powers, followed, after brief and unconvincing effort, by the expression, "Oh, well! an explorative incision will settle it."

Necessarily, the patient is the embodiment of the information on which the diagnostician must depend for his differential conclusions. Here, as in other fields of interrogatory endeavor, only carefully considered plans of attaining a comprehensive knowledge of facts relating to a patient, thoughtfully, courteously, and consistently employed, will satisfactorily accomplish the purpose. The diagnostic efforts of the surgeon should be exercised in all respects in such a manner as will secure frank and unreserved concurrence on the part of the patient. In the absence of a gracious and sympathetic method of inquiry, comparatively little progress will follow the best-planned endeavors of attaining the requisite knowledge. Arbitrary, unsympathetic, and indelicate expression or manner is likely at once to inhibit all concurrent action of the patient related to diagnosis. The patient's and the friends' understanding of the direct and comparative value of testimony in the history of a case is necessarily crude, often misjudged and misleading, and frequently of little practical significance. Yet the earnest desire to impart information which these efforts betoken should be given proper respect, duly emphasized by the pleasant and patient bearing of the surgeon. It will not infrequently happen, for good reasons perhaps, that a patient will decline or evade answering queries derogatory to his own sense of dignity or self-respect, or to his ideas of propriety, or that may intrude on personal secrets or a sense of duty to himself or to another. These sentiments should be respected by the surgeon, who, without appearing overinsistent, may, notwithstanding these obstacles, be able to approximate the truth sufficiently to meet the aims in view.

In all matters of expediency relating to the questioning of patients, due heed should be given to their standards of intelligence and of culture, their sense of refinement, and their familiarity with unsavory and uncanny topics and associations. Some patients, for reasons difficult to explain, will, after being discreetly humored by the surgeon, freely disclose things of signal importance which were at first retained with tenacious reserve. The most delicate form of expression and manner, untainted with any irrelevant references or needless allusions, are requisite in developing the facts of a case in the female sex, especially if they be of a strictly personal nature and in any way encroach on a high standard of proper inherent female propriety. And especially is this plan of action necessary in instances of the young and the unsophisticated of the female sex, and with those whose disinclination to co-operate in an effective manner needs the supporting presence and encouragement of a third person, such as an old friend, a near relative, and sometimes the mother of the patient. Again, sensitively attuned patients of either sex will not infrequently divulge important or perturbing facts more freely and fully in the absence of a third party, especially when the information imparted tends in any way to detract from the dignity, self-respect, or standing of the patient.

The needless exposure of the person of a patient of either sex, or unnecessary dalliance of any sort, as in the use of instruments or in physical examination, or by superfluous and irrelevant talk, especially of a familiar character, should be sedulously avoided. In instances of special examinations of female patients of instrumental or of oral kind, the near-to-hand presence of a third person of responsible station should be had; and, on occasions of the administration of anæsthetics for diagnostic or other purposes, the third person should be present in the room.

It should be remembered that patients differ naturally from one another in many respects, notably in constitutional characteristics, in idiosyncrasies, methods of expression, manner of bearing inflictions of various kinds and of estimating the severity of pain and other manifestations of injury and disease. In other words, each patient, until a different course is determined upon, should be regarded as a more or less independent factor in diagnostic endeavor, and be estimated accordingly in all essential particulars.

The use, on the part of the surgeon, of ambiguous and technical expressions should be avoided, and only such terms should be employed in diagnostic effort as are of easy comprehension by patients already more or less perturbed by the situation and by the fear of the announcement, by the surgeon, of unfavorable findings. The unlettered often mistake the application of common words or may be ignorant of their existence. Hence, if great care be not exercised under these circumstances by the surgeon in taking the history of the case, he will be so misled as to negative an important proposition in diagnosis. "Have you ever been injured before?" is often promptly answered by the patient in

the negative; and, if the answer be as promptly accepted, it will, in many instances, destroy the previous history of the case in this important respect. The incorrectness of the answer is due to the forgetfulness of the patient, or to a failure to appreciate the import of the inquiry or possibly the meaning of the word injury itself. In the taking of the history of a case, it is usually a better plan to permit the patient to make a preliminary statement of his case, guided somewhat by the surgeon, if need be, in order to bring out the logical sequence of events. This plan of action lends courage and gives importance to the patient, and, when advantageously used by him, hastens a correct understanding of the truth, especially when the landmarks thus established by the narrative are utilized by the surgeon in securing a more detailed expression of the facts.

Only rarely, indeed, does one meet with a patient who is unable to give an intelligent account of his own case, even when aided by the inquiring surgeon. In such exceptional cases the surgeon can hope to secure sufficient data for the formation of an enlightened opinion only by paying the closest attention to the patient's statements and by exercising much patience and forbearance. Also it should be remembered that the correctness of a diagnosis and the promptness with which it is secured depend, not only on the complete and accurate history of the case in all respects, but also on the experience, the knowledge, and the sagacity of the surgeon. It is not sufficient merely to give the proper name to the disease, but the extent and location of the tissue changes, the causes, the present and prospective complications, the treatment, the prognosis, and the sequels should each be given due weight in the judgment of the surgeon, based on intelligent appreciation of the information gained from the testimony of the afflicted witness.

Surgeons of large experience are very cautious about utilizing the elements of probability when they come to frame a diagnosis, but the beginners, the impatient, and those whose resources are still undeveloped and who have yet to feel the sting of frequent and mortifying failure are very apt to make an unsafe use of these elements and to draw hasty conclusions. The overconfidence and inattention born of extended experience, of youthful enthusiasm, and of pretentious ignorance are certain to lead with distressing frequency to ignominious failure in diagnosis. It is only those who have properly trained minds and who are willing to gain experience by honest and painstaking labor who can expect to attain eminence as diagnosticians.

Finally, a word of caution should be added regarding the temptation to make long or distressing examinations of patients who are severely afflicted with weakness or pain, with no other object in view than that of making a prompt diagnosis. Nor should the mistaken zeal of the physician or surgeon lead him to sacrifice in the slightest degree a patient's chance of recovery for the purpose of forestalling the autopsy findings.

As before remarked, "The patient is the embodiment of the information on

which the diagnostician must depend for his differential conclusions." The oral testimony of the patient and that obtained by careful systematic scrutiny of his person and of the fluids of his body, together with the knowledge gained by the surgeon through a proper interpretation of the circumstances relating to the case, should, in the great majority of instances, provide sufficient evidence for the determining of a diagnosis.

THE EXAMINATION OF THE PATIENT.

In examining a patient for diagnostic purposes certain well-established common facts, such as the name, age, occupation, habits, family history, etc., of the patient should be given precedence for apparent reasons. Afterward, the line of inquiry may begin with an analysis of the first onset of the disease sustained by the patient, or with that of the present attack. If the former course be adopted, it will be desirable also to investigate the facts relating to all subsequent attacks. This plan may be denominated the direct or the *analytic method* of procedure. The reverse of this practice may be employed by commencing the examination with the present phenomena, and following them back to the beginning of the affliction. This latter method is denominated the *synthetic method* of procedure. We have no hesitation in expressing a strong preference in favor of the former method, since our long experience with it has amply justified this conclusion. And more especially are its advantages apparent in complicated cases and those with long and varied histories of disease or injury. In instances only of recent injury or disease, the briefer plan of analytic examination may be utilized at first, but with the idea of later estimating the effect of remoter troubles on the patient's welfare. The direct or analytic method of examination places before the surgeon in a consecutive and logical manner a complete general history of a patient, and any omissions in this respect will be due to the lack of experience of the examiner or the failure of the patient properly to comprehend the meaning of the questions submitted for reply. In the indirect or synthetic method of examination there is much liability of overlooking important contributive facts, and, too, the method is apt to be more embarrassing to the patient and perplexing to the surgeon than is the analytic plan. However, in instances of local injury and in other afflictions with brief histories, as well as in those requiring prompt therapeutic action, the synthetic plan is usually preferable. As before stated, by means of the analytic plan the facts of a case are gradually revealed in a logical systematic manner, disclosing in a direct way their mutual, consecutive relationship with each other up to the last moment. A record of events of any kind, made from the beginning to the finish, is much more likely to be complete and effectively connected than when the synthetic method is employed. In either instance, however, only great care, fortified by a thoughtful and painstaking method

of making such inquiries, will enable the surgeon to secure a complete and reliable history of a case.

Too often the finding of an unusual or striking feature in the history of a case will cause the examiner to lose sight of the main purpose in view, and follow enticing developments, of little or no contributive importance, to obscure and irrelevant endings. When, in the course of the taking of a history, especially of a complex nature, an unexpected or unusual feature appears, the fact should be specially noted and reserved for later analysis, and not permitted to divert the course of the examination from the regular line of procedure.

The Manner of Questioning Patients.—Already much has been said regarding the general manner of the examiner toward the patient, but it still remains to speak definitely of the arts of phrasing and so systematizing the questions as to make the best use of opportunity. The query commonly addressed by us to a patient is, "Up to what time (year) of your life were you perfectly well?" or, "Were you ever ill or injured? If so, what was the date of your first injury or illness?" After ascertaining by careful analysis the causes, nature, severity, results, and other important features of the first of the afflictions, the remaining ones should be given a similar consideration. The asking of a patient, "What is the matter with you?" or, "What is your complaint?" often amuses the patient and sometimes misleads the surgeon, causing the former to reply, with comical or sententious mien, "I came to you to find out," or to express gravely a diagnosis that has not a good foundation. By the former reply one may be annoyed; by the latter, one is often deceived. Therefore, we have long since ceased seriously to propound these questions, limiting ourselves in this respect to those admitting of no cavil or misunderstanding, such as "Of what do you complain?" "How long have you been ill?" or, "How long ago were you injured?" etc. These are entirely proper queries, calling for prompt and intelligent replies, leading to direct and logical conclusions. In instances of traumatic violence, especially in those depending on a fall or a blow, the facts relating to the cause and the distance of the fall, the manner of striking, and the physical characteristics of the object struck, together with the immediate effect on the ability of the patient to care for himself and the causes of the hinderance to do so, are matters of great importance that should be ascertained at the outset in a systematic, logical manner. So far as it is possible to do this, an estimate should be made of the results likely to follow a blow of a given force.

The Circumstances of a Case.—It is plain that it is of great importance, as regards both the diagnosis and the prognosis, that all the circumstances of a case should be ascertained in the most complete manner; and, in addition to what can be learned by questioning, there should be a most careful examination of the organs and the fluids of the patient, with the idea of disclosing any subtle or obscure threatenings of life. The securing and the proper grouping

of the evidence gained by all examinations, up to the time when the surgeon is called in, constitute the history of the case; and it is proper to say in this connection that a well-taken and wisely comprehended history constitutes the true route to correct diagnosis and rational treatment.

Two methods of examination of a patient are commonly practised—the general and the special method. The former method relates to information regarding the influence of age, sex, habits, occupation, family history, etc., on the afflicted patient. This knowledge is gained by questioning the patient and the friends and relatives. The special method relates to information gained by the personal examination of the patient by the surgeon; it is also often called the special examination. It is manifest that both general and special examinations are absolutely essential to the securing of reliable conclusions.

The General Examination of a Patient.

The Age.—The age of a patient exercises a striking influence on the nature, the effect, the results, and correspondingly on the outcome of treatment, of disease or injury. While youth is decidedly sensitive to shock and pain and loss of blood, yet it is largely exempt from the weakening influences of the responsibilities, acts, and duties incident to advancing age. The subtle effects of physical and mental strain, of deteriorating practices, and the natural changes of advancing years lessen the resisting power of the human organism in almost a direct proportion to their degree and extent. For these reasons children who escape the effects of shock, loss of blood, severe pain, and restlessness recover in an astonishing manner from injury and disease that often promptly terminate existence in adult life. Adult patients differ in endurance from one another more than do the young. Aged patients with good muscular and mental vigor, well nourished but not adipose, having good digestion, sound organs, and pliable vessels, can withstand well the trials of physical infliction and may be classed as enduring patients. Patients, however, with conditions the reverse of these, especially when complicated with alcoholic influences, should be treated with conservative deference, as they often promptly succumb to the effects of even comparatively moderate physical punishment or to the delirium of previous alcoholic excess. The common manifestations of disease in different parts of the body have different meanings at different ages.

A pain in the knee of a child suggests disease of the hip; in the adult, disease of the knee itself. A pain in the bladder of a child may point to stone in the organ, but never to enlarged prostate. Tumors having similar characteristics and locations in the young and the old have different natures and meanings; in the former they are, as a rule, innocent growths; in the latter malignant. Enlargements of lymph nodes in the young are common, often indicating simple irritation or tuberculous infection; in the adult such enlargements are infre-

quent and often dependent on malignant changes. Injury near a joint in the young may cause diastasis, but never in the adult, for manifest reasons. Injuries of equal force are more liable to cause fracture in the adult than in a young person, since the bones break the easier in the latter class. Cutaneous diseases in children are strongly suggestive of one of the exanthemata; in adult life they more commonly indicate other forms of infliction.

The Sex.—Women withstand operations and injuries rather better than do men, a difference due in part to the greater patience and fortitude of the former, increased by the benefit of greater temperance and discretion in things that so often unfit the opposite sex for the patient support of physical hardship. And, too, confinement in bed and general inactivity are less irksome to the female than to the male sex. The physical and psychical natures of the sexes are radically different in health, and correspondingly diverse in disease. The emotional element dominates the female; the physical and the unemotional dominate the male. We find, therefore in the female not infrequently general hysterical manifestations, and also local ones referable to a joint, a limb, the bladder, a special organ, the special senses, etc.—in fact, to almost any part of the body, and, too, presenting rational, or unreasonable, and even grotesque characteristics. Later, these hysterical patients often suffer from an almost omnipresent and depressing fear of cancer of the uterus or breast, even to the end of life. It rarely happens, however, that one of the male sex exhibits hysterical manifestations of a general or local nature. Commonly, local evidence of disease in the male sex justifies the belief that such disease actually exists. The male sex, like the female, has its fears of impending dangers, but of a different nature. In youth and during advancing manhood, fear of heart disease is apt to be the uppermost thought in the patient's mind, followed by a long period of comparative mental rest in this respect. In advanced years, however, he is likely again to be disturbed by apprehensions of enlarging prostate and of cerebral apoplexy. In either sex in these circumstances the motto of the surgeon should be, "Carefully examine, promptly diagnosticate, and quickly remedy a real or imaginary infliction." It is proper to say at this time that the inflictions of the male sex are largely the heritage of occupation, of exposure, of mental and physical hyper-activity, and of bad habits; those of the female, on the other hand, grow out of the complications and sequels of menstruation and child-bearing, combined with those begotten of inactivity, introspection, and emotional domination.

The Occupation.—The occupation of a patient has, indeed, very much to do with the nature, the severity, and the outcome of disease or injury. The occupation itself may directly cause infliction or contribute the influences that favor its occurrence or development. Necrosis of the jaw, lead colic, soot-cancer of the scrotum, patella bursitis, olecranon bursitis, malignant pustule, glanders, etc., are each striking examples of the direct influence of occupation

on physical ills of the watchmaker, the painter and the plumber, the chimney-sweep, the scrub-woman, the miner, the tanner or the wool-sorter, and the stableman, respectively. The modern chauffeur contributes by his calling, as do his patrons by their presence, a fair share, illustrative of the relationship between injury, on the one hand, and occupation and pastime on the other. The "glass arm," the rounded shoulders, the curved spine, etc., testify respectively that the baseball pitcher, the tailor, the shoemaker, and the farmer bear, each one, the indelible stamp of his calling. Those whose avocations expose them to the allurements of overeating and drinking and of late hours, to say nothing of the besetments of vice that often attend such forms of business, are, in many instances, illy equipped to withstand even minor degrees of injury or disease, and they can scarcely hope to recover from injury or disease that in a decided degree imperils the lives of those who have in the past given due heed to personal welfare.

The Habits.—Whether or not the patient has been temperate, virtuous, and law-abiding is of great significance in estimating the probable results in instances of grave injury or disease. The liability on the part of those who indulge in excessive eating or drinking, of contracting a disease, or of directly or indirectly fostering its development by such habits, is of vicious import in lessening human vitality. The manner of dress, the habits of labor, the periods and methods of recreation, the amount and the character of the food and the regularity of eating, along with the use of narcotics, are matters that exercise an influence for good or for evil on the patient, usually in direct proportion to the excess of indulgence. Often, however, for reasons difficult clearly to define, what are apparently indulgences for one person will promptly bring to grief another less immune than he to their effects. It is important to remember that the virtuous may bear innocently and unsuspectedly the evidence of impure associations and their sequels. In such cases as these the surgeon should exercise great discretion; otherwise irremediable sorrow and perhaps unmerited disgrace will be unwisely added to distressing affliction.

The Antecedent History.—The antecedent history of a case should include, not only the past record of the patient in all matters relating to disease or to injury, but also, when the question of disease is involved, that of his ancestors. Either a special or a general invulnerability to disease on the part of a forebear may be transmitted, and when this happens it may manifest itself primarily in the first generation, sometimes in the second, and perhaps even in a later generation. According to Colles, syphilis of the child means syphilis of the mother, whether or not the mother shows other symptoms of the disease. The child, however, will infect the nurse. It not infrequently occurs that a young patient with pronounced or scanty manifestations of glandular, nervous, ocular, auditory, or other symptons of disease is allied remotely or immediately with unsound ancestry bearing a history strongly suggestive of syphilitic or

tuberculous infection. In such cases as these, especially those suspected of a syphilitic taint, wise discretion should prompt the surgeon to employ the requisite treatment without arousing incriminating suspicion on the part of those who may regard with justifiable pride the praiseworthy records of an honored ancestry. The recognition of certain family characteristics compels the belief that rheumatism, gout, hæmophilia, color-blindness, tumors of a simple or of a malignant nature, are apt to be transmitted from parents to their offspring. That this should be so is emphasized by the well-known facts of the transmission of personal distinguishing traits from parents to children. In view of the fact that there are numberless instances in which the transmission of such diseases has not taken place, we should be admonished not to give too great heed to coincident marked disease in those who bear a blood relationship to the patient. The antecedent history of a patient with reference to disease and injury falls better for consideration under the personal history of the patient.

The Personal History.—The personal history of a patient should include a record of his personal characteristics as well as of the diseases and injuries which he has sustained, and of their outcome. Incidentally, his habits, more especially the objectionable ones, are subject to review, since they may have a very important bearing on the prognosis and treatment of the patient. The temperament of a patient has not a little to do with the outcome of surgical injury and of disease, as well as with the results of operative interference. The patient with a full pulse, vigorous heart, high arterial tension, warm surface, and excitable nature, is more liable to unfavorable reaction from injury and surgical effort than is one with the reverse characteristics. A patient with sluggishness of thought and action, and comparatively indifferent to suffering and confinement, usually bears well the inflictions of physical injury. As a general proposition, those of good physical vigor bear operations better than those of a feeble state. However, the athlete who prides himself on his strength of frame and fleetness of limb, whose entire system is fitted only for active effort, is unsuited for the confinement of the sick-room. The semi-invalid and the one to whom confinement and inactivity bring no special regret are, other things being equal, better fitted for the ordeals of an operation than is a trained athlete. When expedient, therefore, the former class should serve a brief preparatory period in confinement, attended with free unloading of the system by the emunctories, before an operation is commenced.

The obese patient is ill fitted for an operation, especially when the obesity is the result of indolence, luxury, or intemperance. Hereditary obesity is of less moment than is the acquired, especially when it occurs in a person who has not been able to exercise control over his appetite. Physiologic plethora, when present in a person who is otherwise physically and functionally vigorous, offers no obstacle to recovery from injury or disease, or to the securing of successful results from operative effort; but the acquired plethora of the tippler

and the gourmand should be as a beacon warning against operative practices not supported by the logic of expediency and not protected by the strictest modern technique. In the presence of an obscure or incomprehensible injury of a patient, apparently due to assault, it is a relief to know that it may be dependent on the effects of alcohol or epilepsy. On the other hand, a knowledge of the fact that a person is a victim of epilepsy may spare him the ignominy of being regarded as the slave of intoxicants. In making these estimates, however, it must be remembered that a liquor-laden breath does not surely indicate the habitual use of alcoholic drinks; some solicitous friend or some good Samaritan may have given liquor as a remedial measure to a patient who had never before experienced its taste.

The deformities incident to a previous injury of a patient, especially of the skull or a joint, are of significant import in estimating the gravity of a recent injury located at the seat of, or involving the functions of, the part previously injured. The history of a previous fracture of the hip or thigh, with or without shortening, in the presence of a recent severe injury of these parts, is of immense importance, and such knowledge may be absolutely necessary for determining the degree and the extent of the present injury.

The great lesson taught by the preceding facts is: Carefully take the history of the case if you expect to make a correct diagnosis. Whether or not a patient be single or married, happy or unhappy, active or idle, whether sexual indulgence be occasional or frequent, lawful or illicit, are each a matter worthy of careful scrutiny in either sex. The history of the effects of menstruation, child-bearing, miscarriages, and the complications and sequels of parturition on a patient ought to be carefully weighed.

The environment of a patient is a matter of great importance, since those who are favored with healthy surroundings are much the better fitted to meet the contingencies of injury and operative practice. Patients who are exposed to the deteriorating influences of special miasms, bad ventilation, damp and sunless surroundings, are poorly qualified to meet physical emergencies, especially when the quantity, quality, and amount of food partaken fall short of the natural demand. Mental emotions of a depressing nature, such as apprehension, fear, remorse, disappointment, etc., from whatever cause, particularly when of a direct personal bearing, as fear of the outcome, whether real or imaginary, exercise discouraging effects on many patients. Steadfast hope, sure and abiding faith in the medical attendant, aided by a genial and philosophical nature and encouraging associations, contribute more to a successful issue in many cases than the faithful utilization of the therapeutic agents of a near-by pharmacist. The therapy of hope wields a mighty influence in recovery, and ought always to be administered with a free hand when circumstances justify such a course.

The nature of a morbid growth may be estimated, with considerable proba-

bility that the estimate will prove to be correct, by the length of time that it has existed, for if of long standing, with no special evidence of malignancy, it may be regarded for the time as an innocent growth. On the other hand, a rapidly increasing local growth, attended with local discomforting symptoms, should not be regarded as a harmless development, but should be promptly suspected to be of a malignant or destructive nature until otherwise determined.

It appears proper at this time to assert that the nature of a growing tumor should be investigated at once, and that it should be promptly removed when the least suspicion of malignancy is found.

Enough has been said already to emphasize the great importance of a careful general examination of a patient for diagnostic purposes. Hardly less than this would suffice, and not more can be said here because of the limited room allotted to this article.

The Special Examination.

The special examination includes the physical, both being of a general character. The examination of a patient may be conducted to a final conclusion by the combined diagnostic products of the following fields of inquiry:

1. Inquiry by the unaided senses of sight, touch, and hearing, and sometimes of smell.

2. Inquiry addressed to the digestive, respiratory, circulatory, nervous, locomotor, and genito-urinary systems.

3. Inquiry directed to examination of the secretions, the excretions, the discharges, and other fluids of the body not already considered. (See the article next in regular order.)

4. Inquiry facilitated by the use of anæsthetics and by drugs of narcotic effect; also inquiry supplemented by the findings of explorative and operative aid.

Simple Inspection.—An experienced and observing surgeon can quickly determine the nature of certain diseases and injuries by noting the *posture* of the patient or of the injured part. The striking signs of coxitis, of dislocation of the head of the femur, and of fracture of the femur are such as to make a diagnosis of one from the other, by means of inspection, not at all difficult under ordinary circumstances. The dorsal posture of a patient with flexed limbs, the distended abdomen, the thoracic breathing, and the anxious facies suggest so decidedly the presence of peritonitis as to require strong opposing evidence to effect a change of opinion. A patient carrying flexed an injured arm, supported by the opposite hand, with the head inclined to the injured side, will quite likely have sustained a fracture of the clavicle. A young patient, who in walking carries the body straight and stiff, with the shoulders elevated; who moves with a shuffling gait, stepping down with deliberation and care; and

who, on standing, leans for support in an involuntary manner on friendly objects, presents a familiar picture of spinal caries, which is completed beyond gainsay when the patient squats instead of bending forward to pick an object off the ground or floor. A patient with severe injury of the neck, who, with anxious facies, on moving grasps firmly the head between the hands, rigidly holding it thus while turning the body to either side for any purpose, may have fracture or dislocation of the cervical spine. The sitting posture betokens oppressive breathing, more especially when the shoulders are fixed by contact of the upper extremities with an unyielding support. Involuntary sliding down in beds points to extreme exhaustion. A patient with acute pleurisy from traumatism or disease, or one with pneumonia from the same cause, will lie upon the afflicted side so as to limit painful motion. In pleuritic effusion, and sometimes in thoracic aneurism and in movable abdominal tumors, the patient will lie on the diseased side to gain the increased comfort afforded by unhindered expansion of the unaffected side. In severe colic from whatever cause patients usually lie in the lateral position, with the limbs and body flexed on each other; then again, in severe abdominal colic, in gastric ulcer, in aneurism, and in vertebral caries patients lie on the abdomen to secure the relief afforded by direct pressure, as well as that which comes from a change in the position of the spine and of the abdominal contents. The color of the skin, the state of nutrition of the body, the degree of muscular development, etc., are each important, as indicating whether or not the patient be temperate or anæmic, or be suffering from disease of the heart, the liver, or some other organ.

The superficial or pictorial anatomical appearances of definite parts of the surface of the body, as related to injury and disease, present diagnostic factors often of great value. In order properly to estimate the diagnostic value of abnormalities in appearance, one should be familiar with the normal surface outlines. Owing to the differences which naturally exist in certain regions of the body in health, a proper estimate of the changes following injury or disease of a part is often made with difficulty.

The Surface of the Body.—In examining the surface of the body, it is especially essential that the patient be placed in a good light which shall fall with equal intensity upon corresponding portions of the surface, *i.e.*, the abnormal and the normal. The patient should lie straight, or sit erect if practicable, with the limbs placed symmetrically. The influence of respiration on the symmetry and movements of the thorax and abdomen should receive close attention. Due allowance should be made for the effects of an unequal shedding of light on a part and for any abnormal coloring of the skin dependent on disease or upon some artifically colored rays of light.

The flattening of the shoulders due to fracture of the acromion process, to dislocation of the head of the humerus, or to atrophy of the deltoid, is very liable to cause—even in the mind of an experienced surgeon—perplexing doubt

as to the true state of affairs. The outline of the spines of the vertebral column and the prominence of special ones are matters of prime importance, as undue deviations of the former and increased prominence of the latter bespeak a marked degree of lateral curvature and of antero-posterior curvature (Pott's disease), respectively. If we wish to render more evident the degree of lateral deviation of one or more of the vertebral spines, it is only necessary to rub the skin with the fingers several times, with some degree of force, over these bony prominences. The skin at these points will thus be rendered red.

Inspection of the Thorax.—Inspection of the thorax enables one to note whether or not the intercostal spaces and the respective sides of the chest respond normally to the respiratory acts, thus determining the presence or absence, in the chest, of fluid and its location. Such an inspection will, at the same time, reveal to us whether or not the heart be much enlarged or unduly active, and whether the thorax be symmetrical. The presence of collapsed lung or of fractured ribs causes restrained respiratory movements.

Inspection of the Neck.—The two sides of the anterior aspect of the neck should be carefully compared, so that one may be able to note, later, the slightest abnormality of outline or of action. The fact that malignant disease, aneurism, glandular tumors, bronchocele, etc., are not infrequently located in this intricately constructed and important region sufficiently emphasizes the need for a most careful examination of the parts. In gunshot or stab wounds of this region emphysema of the subcutaneous tissue of the neck should lead to prompt investigation of the integrity of the trachea, the œsophagus, and the lung. The circumscribed areas lying directly beneath the lobes of the ear are specially worthy of study, since here is often seen the earliest evidence of enlargement of the parotid gland and of the overlying lymph nodes.

Inspection of the Face.—The distressing tetanic grin, the pinched features, and the gasping inspiration of approaching dissolution, the involuntary frown of peritonitis, and the facies which expresses apprehension of the torturing spasms so frequent in acute disease of joints and in severe neuralgias, notably of the trifacial type, are of common occurrence. A knowledge of the relationship of the eyes to their bony environment and to each other enables the surgeon to discover, comparatively early, the presence, in the antrum, of a rapidly increasing growth, encroaching on the cavity of the orbit and its contents, and consequently to adopt the necessary surgical measures before the disease has advanced too far.

The Abdomen.—The surface of the abdomen should be inspected with care, so that abnormal deviations referable to the outline, color, markings, movements, and the circulation may be quickly noticed and their significance readily estimated. For example, it is important, in making the distinction between a hernia, on the one hand, and a hydrocele or a scrotal tumor, on the other, to note whether or not an inguinal tumor first appears from above or from below,

or remains *in situ* when the patient in standing or when he is lying down; and whether it does or does not convey an impulse on coughing. Even more important in many respects is a similar scrutiny of the femoral areas, for here hernia, abscess, glandular growths of simple, specific, and malignant natures find an open door, and, unfortunately, their presence and nature are in many instances ascertained too late to permit of the administering of satisfactory relief. The lymph nodes of this situation freely communicate with near-by clusters of similar nodes, offering early opportunity for extensive infection. Inspection, especially of individuals with thin abdominal walls, reveals the presence of intra-abdominal tumors of considerable size, and also, in some cases, the increased peristalsis characteristic of intestinal obstruction.

The Color.—In the diagnosis of disease and injury color plays a part of considerable importance. The scarlet blush of acute inflammation, the dusky red of subacute inflammation of a complicated or of a specific character, the mottled hues of venous obstruction and the livid one of asphyxia, the varying shades of color observed in traumatism, the inky aspect of dying tissue, the uncanny pallor of anasarca, and the sallow and waxy hues of advanced malignancy—all of these testify in some measure to the important information which color may convey.

Translucency of a diseased part can be quite well considered in connection with color. A translucent tumor is largely made up of a thin, colorless fluid, as in hydrocele of the scrotum and of the spermatic cord; also in spina bifida. The normal tissues of the hand, the ear, and the cheeks are, in thin persons, when subjected to a powerful light, fitted to the purpose.

Palpation.—On the abdomen, more particularly than elsewhere, palpation should be practised with gentleness and care, especially when the part thus examined is tender or inflamed or liable to be bruised or ruptured by the act. Incautious palpation of a diseased appendix, or of an abcess or cyst, may cause prompt rupture, with fatal extravasation of the contents. In palpating, the hand should be warm, be laid flat on the surface, and be allowed to remain quiet until the patient and the part are reconciled to its presence. Circular or to-and-fro movements, made in opposite directions and gradually increased in force and area, are employed to determine the depth, the sensitiveness, and the mobility of the deeper parts and of the overlying tissues. If the movements are too vigorous or the ends of the fingers are carelessly used for the purpose at first, the aims of the measure will be defeated by the muscular contractions due to pain, to acute expectancy of the patient, and perhaps to irrational objections as well. The cautious and deliberate use of the ends of the fingers in palpation enables one to judge of the size, the depth, the mobility, and the physical characteristics (hard, soft, elastic, irregular, etc.) of a growth, to say nothing of the degree of sensitiveness of the parts. By palpation we determine the presence of fluctuation due to the existence of fluid in the tissues. Muscular

fluctuation may be mistaken for that caused by the presence of fluid, unless the muscles be palpated in the long axis instead of the transverse, when the fallacy will disappear.

The crackling of emphysema, the crepitus of fractures, the thrill of an aneurism, the friction of roughened synovial and serous membranes, and the creaking of joints, etc., are easily determined by means of palpation. The weight of a morbid growth springing from a pendulous part of the body, like the mammary glands, or the testis, or that of a pendulous growth elsewhere located, is determined by a sense related to that of touch; and since malignant and fibrous growths are denser than those of a fatty or cystic nature, this element of dissimilarity may be of use in differentiating them. An estimate of the temperature of a part, like the estimate of its weight, can, in many instances, be made by touch sufficiently well for all practical purposes. The fallacies, however, that may arise in this practice are numerous. Thermometers are so universally available, and their importance as an aid to diagnosis is so thoroughly established, that one is not justified in placing more than a passing reliance on a mere manual estimate of the temperature of a patient.

The Examination of the Principal Systems of the Body.

The principal systems of the body, as arranged for the purposes of our present study, are five in number, viz., the digestive, the respiratory, the circulatory, the nervous, and the genito-urinary systems. But with the limited amount of space at our disposal, we can scarcely hope to do more than touch very lightly upon the questions of diagnosis as they are related to each of these systems.

The Digestive System.—The digestive system comes first in the natural order of distribution, and it also deserves that position on account of its commanding importance.

The Lips.—The smooth lips of the young, the pallid lips of the feeble, and the cyanosed lips of those with deficient aëration of the blood are pictures with a significant meaning. The unclosed lips of dyspnœa, when parched and purple, indicate acute or chronic interference with the proper oxygenation of the blood. A downward and outward deviation of one angle of the mouth may be due to loss of power on the opposite side, or to undue contraction on the same side, as in facial paralysis and in cicatricial contraction respectively. The swelling of a lip may suggest traumatic violence, deep-seated inflammation, great local irritation, the urticaria of idiosyncrasy, the bite of an insect, or the effect of an injury inflicted during epileptic convulsions. A fissure, an ulcer, or a mucous patch of the lip are lesions which suggest a specific nature. An ulcer of the lip with induration of the related lymph nodes may be of either a malignant or a specific nature, and the microscope should be employed to determine the question.

The Gums.—The swollen and spongy gums of scurvy, the blue line of lead poisoning, the red line of tuberculous and of cancerous cachexia and of diabetes, and the pallor of anæmia are among the many indications of disease related to the gums.

The Teeth.—The early dentition of precocity and of syphilitic endowment, the delayed dentition of rickets and cretinism, the peg-shaped, notched teeth of syphilitic belonging, the teeth with dentated edges and furrowed surfaces of bad nutrition, the loosened and decayed teeth of scurvy, of purpura, and of phosphorus poisoning, are manifestations of great diagnostic value.

The Tongue.—The tremulous tongue of the alcoholic, the halting protrusion dependent partly upon loss of muscular power and partly upon mental obtuseness, the genuine deviations caused by organic paralysis, the apparent deviations due to facial paralysis, are of diagnostic importance. The smoker's patch, the syphilitic mucous patch, the chancrous, the tuberculous, and the malignant ulcers should each be promptly recognized and their meaning estimated. The dry tongue of mouth-breathing, of continuous high temperature, of dehydration of the body, of asthma, of prostration, and of mental emotions is in each instance significant.

The Palate, Tonsils, and Pharynx.—The offensive odor of follicular tonsillitis, of gangrene, of cancerous and syphilitic ulcerations should be noted. The discoloration of the palate or pharynx due to venous obstruction, and the presence in these situations of circumscribed hemorrhagic points, with or without the escape of blood, require investigation. Perforations or ulcerations of the hard or the soft palate and adhesions of the latter to the posterior pharyngeal wall point to syphilis. Bilateral paralysis of the soft palate suggests diphtheria, possibly vertebral caries; unilateral denotes deep-seated interference, as in intervertebral pressure and fracture of the base of the skull. An aneurism in close proximity to the faucial tonsil may cause the latter to pulsate. Deep-seated ulceration of the tonsil suggests cancerous or syphilitic invasion that may involve the internal carotid artery and cause prompt death from hemorrhage. Acute tonsillitis attended by deep or superficial suppuration, especially the former, displaces the tonsil toward the median line, and calls for an incision to afford prompt relief.

In exceptional cases the tonsil and contiguous tissues may be the seat of an extensive invasion of malignant disease which nevertheless causes so little local disturbance as to fail to arouse a suspicion of serious trouble.

The mucous membrane of the pharynx is liable to various phases of disease of simple, specific, or malignant nature; it is also subject to acute and chronic expressions of the various forms of pharyngitis. The pharynx may be encroached upon by a post-pharyngeal abscess, causing slow or rapid interference with deglutition and respiration; such an abscess is due to vertebral caries or to some deep-seated infection close at hand or more distantly located. Difficulty

in swallowing may also result from rheumatic lameness, from spasm, from paralysis of the muscles of the larynx, or from hydrophobia, tetanus, or strychnine poisoning.

The Œsophagus.—Stiffness of the neck due to acute inflammation of the œsophagus or to pre-œsophageal suppuration simulates that which is dependent upon traumatism or upon rheumatic invasion. Diseases invading the mcuous membrane of the œsophagus are characterized by the production of a glairy and sometimes frothy or viscid mucous secretion, which is dislodged by hawking, and is especially abundant in acute inflammation and cancer of the gullet. Hemorrhage of the œsophagus usually comes from the lower end of that tube, being due to varicose veins or to obstructive disease of the liver or heart, especially in old people; it also may come from intra-mediastinal pressure, from cancer and other forms of ulcer, and also from the presence of a foreign body or from a severy injury. The amount of blood discharged is usually small, alkaline, of bright color, and not mixed with the contents of the stomach unless vomiting has happened. Emphysema in the connective tissues of the neck suggests perforation of a lung or of the trachea or of the œsophagus. In the latter case there must exist a communication between the œsophagus and the trachea or one of the deeper bronchi, such a condition being due in all probability to ulceration or to a wound of these passages. When obstruction exists in the course of the œsophagus, auscultation to the left of the ninth or tenth dorsal vertebra may reveal delayed or absent second sound of normal deglutition. Any pathological change causing local or general enlargement of the œsophagus in the neck can be felt behind the trachea, usually better to the left side. Percussion may disclose conditions corresponding to those which have just been mentioned; and further confirmation may be obtained by the employment of the x-ray, which reveals the presence of a bright area (emptiness) immediately next to a dark one caused by the presence, in the unequal lumen, of a mixture of bismuth or some other metallic salt introduced for the purposes of the test.

The technique and the danger of introducing an œsophageal bougie should be well understood. In this connection I may say that the examiner will do well to refresh his memory regarding the size, direction, length, and other anatomical characteristics of the œsophagus, and also to study well the diseases to which this organ is liable, in order that he may not, by his explorative procedures, expose the patient to any unusual danger.

The Stomach.—The general anatomical relations and characteristics of the stomach cannot properly be stated here. The amount of information that may be gained by an examination from the outside is largely regulated by the degree of adiposity of the abdominal wall and the size of the stomach. Usually, when the patient is lying on his back, it is possible to outline a distended stomach by the surface shadow of the lower border, which moves with

the respiratory act. Visible gastric peristalsis, in which the movement progresses from the left costal arch downward to the right, is an indication of pyloric obstruction; so also is displacement or distention of the organ, conditions which can quite readily be ascertained by the modifications of the normal percussion and succussion areas, etc. By transillumination with appropriate apparatus one may determine, with a fair degree of accuracy, the size, shape, and location of the stomach; also the presence of tumors and such other morbid changes of the viscera and contiguous structures as may modify the transmission of light. This plan of investigation, however, along with that of the x-ray and bismuth test, should supplement rather than supplant the findings of palpation. Palpation and percussion are fertile methods of ascertaining the truth in regard to conditions of the stomach, and when combined they are the most conclusive of all. The method of palpation already noted should be practised here, and its efficiency will be decidedly increased by placing the patient in the knee-elbow position, thus causing the stomach to fall forward upon the abdominal wall. These measures are decidedly enhanced in value by co-operative action on the part of the patient, and the measure of success obtained will depend largely upon the degree to which the abdominal muscles are relaxed, the thinness of the abdominal wall, and the size, mobility, and degree of involvement of the stomach. When all these conditions are unfavorable, and notably when they are associated with pathological changes in neighboring organs, it becomes wellnigh impossible to make a diagnosis. When the palpating fingers meet with increased resistance, especially of an unyielding, hard, and irregular nature, there is substantial ground for suspecting the presence of a cancer, and particularly so when the mass occupies the usual site of a carcinoma of the stomach. Although cancer of the pylorus is commonly felt, as one might expect, above the navel and to the right of the median line, it sometimes occupies a lower position. When the tumor is confined strictly to the pylorus, it is not likely, even though it may be freely movable from side to side, to move up and down to any extent in harmony with the respiratory movements of the diaphragm; but, when it is adherent to some freely movable organ like the liver or the diaphragm, then its movements will correspond to those of the part to which it is adherent. Cancer of the stomach is usually a small, hard mass with an irregular surface, while cancer of the neighboring retroperitoneal lymph nodes presents a broad surface, also hard and nodular, but influenced little or not at all by the respiratory movements.

When palpation in the region of the stomach reveals the existence of an area of diffuse tenderness, varying in severity at different points, one is warranted in concluding that a diffuse gastritis, or perhaps simply a dyspepsia, is present. When the ingestion of hot or stimulating substances, or of certain articles of food, causes pain, and when, further, this pain is aggravated by palpation, it is

permissible to suspect the existence of a gastric ulcer. If such an ulcer really exists, it will be found that cool, demulcent, and soothing drinks lessen the pain. Diffuse pain in the region of the stomach, and pain also in the back, are symptoms of which patients affected with cancer of the stomach sometimes complain. Such spontaneous pains are less common in cases of ulcer of the stomach. Finally, there is a certain number of cases of both forms of disease in which the patient makes no complaint whatever of pain.

Lactic acid is usually, but not always, present in the gastric contents of patients affected with cancer of the stomach.

There are many difficulties in the way of determining, by means of percussion, the boundary lines between the stomach and adjacent organs. When the stomach contains a watery fluid and the colon gas, or *vice versa,* there is no special difficulty in determining the topographical relations of these organs to each other. In the case of a distended stomach the relative positions of the greater curvature and the navel furnish a simple and fairly trustworthy indication of the size of the organ. There is one condition, however, in which this guide cannot be safely followed; I refer to the descent of the stomach without any associated enlargement. Adhesions of the stomach to adjacent structures are very likely to diminish the area which the organ normally occupies, as determined by percussion. On the other hand, when the stomach loses some of its motor power and, as a result, is distended, the dimensions of this area will be increased.

Auscultatory percussion intensifies the sounds and enables the diagnostician to determine with greater precision the outlines of distended organs. The changing of the position of the patient, or the introducing into the stomach of a soft bougie, often adds to the value of a previous estimate.

Auscultation enables us to determine the presence of the deglutition sound of œsophageal obstruction, as already stated above, and of the splashing sounds of fluid in the stomach and even in the transverse colon. The latter may be heard upon simple palpation, with or without the aid of a stethoscope. In some instances distinct splashing can be heard at a distance from the patient, without the aid of any instrument. When, long after eating or drinking, a splashing sound is caused by vigorous palpation, dilatation or atony or displacement of the stomach may be inferred.

The Stomach Contents.—A knowledge of the various test meals employed and of the technique of lavage is essential to a suitable understanding of the diagnosis and treatment of diseases of the stomach. These are matters, however, which belong more strictly in the domain of medicine than in that of surgery, and we will therefore say but very little here on the subject. Free hydrochloric acid with no lactic acid characterizes the products of normal digestion. Conversely, the absence of hydrochloric acid suggests the possible presence of a cancer of the stomach, while the presence of alkaline or offensive vomiting

indicates hemorrhage from the stomach and fecal invasion of the organ. The presence of bile and of various medicines mixed with the contents of the stomach materially changes the appearance of the stomach contents in health and in disease. It is therefore important to weigh this fact carefully before final conclusions are drawn. The presence of a splashing sound in the stomach may be present for two or three hours after the taking of food, especially when a liberal amount of fluid is ingested at the same time. When the splashing is heard at a later period than this, the noise suggests obstructive changes in the stomach, loss of tone, or of delayed absorptive powers. The presence of undigested food in the stomach six or seven hours after ingestion indicates conclusively an abnormal delay in the gastric digestive process, and calls for a careful survey of the case. In this connection it should be noted that vomited and regurgitated ingesta present distinctive differences. In the former, disintegrated muscular fibres are mingled with the characteristic stomach contents; in the latter, the fibres are intact and not associated with gastric matters.

The "stagnation test"—*i.e.*, the determining, by the stomach tube, that the digestive functions of the organ are performed more slowly than they should be—is of great practical utility.

Mucus is freely expelled from the stomach by vomiting in cases of severe gastric irritation and in other gastric disorders, and often, too, it is mingled with the fluids of the œsophagus and the mouth. The employment of the recognized tests for saliva and for mucus is essential under these conditions.

Blood expelled from the mouth by vomiting may have come from the stomach, the œsophagus, or the lungs, or from the mouth or its accessory cavities. The source from which it originates is clearly a matter of great importance. Blood coming from the stomach is expelled by vomiting, and is mixed at first with the gastric contents; later, if the stomach has been emptied and if nothing has been introduced into it since the vomiting occurred, the blood will be found unmixed with food. If the blood be abundant and if it be promptly expelled, it is florid; but if it be meagre in amount or long retained in the organ, or swallowed, the redness is not marked, and the matter vomited may be of "coffee-ground" appearance. When bile becomes mixed, in the stomach, with ingesta of various kinds and with Epsom salts (administered for the purpose of inducing catharsis), there is produced a mass which, when brought to view by the act of vomiting, resembles so closely partly digested blood as to require special means to make a clear distinction between the two. In cancer of the stomach hemorrhage is comparatively frequent, but rarely profuse. In ulcer of the organ the reverse of these manifestations is more often the case. The destructive blood changes of acute or chronic disease, the traumatism produced by external violence or by vomiting, and the engorgement of the blood-vessels due to portal obstruction may each cause hæmatemesis of a trivial or severe character.

Blood from the œsophagus is usually of small amount, unless it be due to

rupture of an aneurism, it is expelled by regurgitation, and is not mixed with the stomach contents except in those cases in which it may have been swallowed and afterward expelled by vomiting. The fluids of the mouth and the œsophagus, and perhaps the secretions of the bronchial tubes, may be mixed together, provided that much irritation of the first two regions has been present or that coughing has attended the escape of blood. This history of bleeding from the œsophagus should contraindicate the use of an œsophageal tube, until a thorough examination has established the fact that the patient's need is greater than the danger attending the employment of the instrument. The presence of an aneurism in immediate contact with the œsophagus should be especially thought of in this connection. Blood from the lungs is expelled by coughing, as a rule, and is not mixed with the contents of the stomach, unless vomiting has been provoked by severe coughing or from some other reason. The amount and color of the blood vary according to the cause of the hemorrhage and the condition of the patient. Bronchial bleeding may be noted in the form of streaks in the mucus expelled by coughing, or it may be quite profuse. In both cases the blood will have a florid color. When the blood comes from pulmonary tissues the amount may be small or profuse, florid or dark, according to the extent to which the disease has invaded the arterial or the venous supply. The rupture of an aneurism into the bronchus suggests the escape of a large amount of blood, although at first it may be only of small amount. In the case of repeated hemorrhages the amount increases rapidly. Blood from the mouth may originate from either of the sources already described, and be detected by the same characteristics as before stated. Blood from the pharynx, the posterior nares, and other parts of the region may be swallowed in considerable amounts, especially while the patient is in the recumbent posture; and, if it be expelled by vomiting, it may be mistaken for gastric hemorrhage—an important fact in fracture of the base of the skull, in which condition it is quite liable to occur because of the semi-conscious state of the patient. At this time it is wise to remind the reader of the general constitutional effects of great loss of blood (shock), and also of the influences, on the blood, of profuse and repeated hemorrhages. (Consult the articles on these subjects in the present volume.)

The Liver.—The relation of the liver to the chest wall, the diaphragm, the peritoneum, the pleura, the intestines, and the gall bladder, together with the modification of those relations incident to the mobility of the organ, furnishes a series of anatomical facts of comprehensive importance in relation to diagnosis. Briefly expressed, the upper limit of liver dulness, in the normal state, corresponds, in the mammillary, midaxillary, and scapular lines, to the sixth, eighth, and tenth ribs respectively. The normal width of liver dulness, in the midsternal, the mammillary, the midaxillary, and the scapular lines, is three and three-fourths, four, six, and three inches respectively. These and associated topographical facts afford an opportunity of estimating the degrees of change

in the general and circumscribed modifications of the area of liver dulness, as caused by disease either of that organ or of contiguous organs. Passive congestion, amyloid disease, leukæmia, hypertrophic cirrhosis, cancerous infiltration, etc., furnish illustrations of the diseases which may cause a general increase in the size of the liver; atrophic cirrhosis and acute yellow atrophy affording the best examples of diseases which may cause a diminution of the size of the organ.

Circumscribed modifications of liver dulness are due to the presence of abscess or cancer or cyst, and to such deformities as floating lobes, Riedel's lobe, and tight-lacing liver, so located as to interrupt the normal outline of liver dulness. Other factors which may alter the area of liver dulness are an enlarged gall bladder, a diaphragmatic hernia, a rickety thorax, and Pott's disease of the spine. Pulmonary emphysema, pleuritic effusion, an intrathoracic tumor, and subphrenic abscess (between liver and diaphragm) are among the diseases that change the superior outlines of liver dulness and at the same time displace that organ downward. On the other hand, the liver may be displaced upward, and the outline of the area of dulness be correspondingly altered, by any of the following pathological conditions: an abdominal tumor, such as a pancreatic cyst or a cyst of some other organ; abdominal distention from the presence of gas in the intestines or of pus or other fluid in the peritoneal cavity; collapse or contraction of the lungs; and paralysis of the diaphragm. Then, again, other areas of dulness—due, for example, to the presence of an accumulation of fæces in the colon or to cancerous or tuberculous disease of the omentum or of the posterior aspect of the kidney—may become blended with that of the liver, and thus greatly enhance the difficulty of making a diagnosis. Finally, it must not be forgotten that, under normal conditions, the liver is moved appreciably downward by the diaphragm with each full inspiratory act. In penetrating wounds of the thorax, it is important to know that the dome of the diaphragm corresponds to the fourth rib on the right and to the fourth interspace on the left side of the chest, and that ordinary respiration alters this curve but slightly, while a full inspiration makes a decided change, as the influence on the line of liver dulness will disclose. In instances of perihepatitis from traumatisms of various kinds, from abscess, and from inflammation of the liver, friction sounds, tenderness, and even enlargement of the organ may be found on physical examination.

The location of the pleural (costo-phrenic) sinus and the relation of the pleura to the diaphragm, and of the liver to this muscle and to contiguous viscera, are each of much significance. Before operating, for the purpose of evacuating an abscess of the posterior portion of the liver, it may be found necessary to obliterate the pleural sinus; the purpose of this preliminary step being to prevent infection of the pleural cavity. On the other hand, it is sometimes found that this obliteration has already taken place through the action of an inflammation of recent or of old date. The obliteration of this sinus cripples

the action of the diaphragm. The diaphragmatic pleura and the opposing pulmonary pleura may unite to protect the lung for a time against the invasion of sub-diaphragmatic or hepatic abscess. The liver may be safely approached from above through the diaphragm by reflecting upward from it the diaphragmatic pleura. Finally, it must not be forgotten that the relation of the gall bladder to the costal end of the ninth (freely movable) rib, and the presence there of the lower border of the liver in health, are anatomical facts which have an important bearing upon diagnosis.

The Intestines.—At the outset one ought to be entirely familiar with the topography of the abdomen and its contents in health; otherwise, even marked abnormal deviations will not be noted and, as a result, their significance will not be appreciated. A reasonable knowledge of the anatomy of this region enables one to understand why it is that, in a distended colon, the distention is well marked except at the splenic and hepatic flexures; why it is that abdominal distention, beginning at the upper or the lower part of the abdomen, indicates, in the former case, distention of the stomach and jejunum, and, in the latter, of the ileum alone; and, finally, why it is that a special tympanitic enlargement at the right iliac fossa tells of a distended cæcum. The latter fact is often of great importance, indicating, as it does, at which side (colonic or enteric) an obstruction exists, and often causing the cæcum to be regarded as the "key" to the seat of intestinal obstruction. The relations of palpation and percussion to the presence of fluid in the abdominal cavity, and the change in the positions of percussion sounds in their relations to one another, due to changes in the position of the patient, ought to be kept clearly in mind, as being important diagnostic facts. When a loop of small intestine is in a distended state, due to a distal obstruction in the immediate vicinity, it is possible, in the case of a person with thin abdominal walls, to excite easily visible peristaltic movements by the application of cold to the abdomen.

A knowledge of the anatomical topography of the abdomen makes it possible for the diagnostician to determine, with a fair degree of accuracy, what pathological changes are taking place in the different parts of the underlying abdominal cavity; and a further knowledge of the relations in health and disease between the gall bladder (distended) and the kidney, and the modifications which respiratory action may cause in these relations, furnishes a safe basis upon which a differential diagnosis may be constructed. In estimating the size, location, and degree of mobility of the kidneys, it will be found that inspection, percussion, and palpation (mainly bimanual), in combination with a careful consideration of all the symptoms and an examination of the urine, furnish us with the only means of arriving at the truth.

Investigations in this field are very difficult to carry out, and require, if successful results are to be attained, considerable skill and experience. The same remarks apply with nearly equal force to the spleen and to other organs

of the abdominal cavity. The consideration of all the details connected with the diagnosis of the different diseases of the abdominal organs does not form a part of the writer's purpose in preparing the present article. These subjects will all be fully discussed in the special articles which are to appear in the later volumes, and the reader is therefore referred to them for any further information which he may desire.

The method of ascertaining the condition of the contents of the pelvis by means of the finger introduced into the rectum or the vagina is one of the most valuable methods of diagnosis that we possess. By it we are enabled to learn the presence of an obstruction in the intestinal canal and to form a fairly good idea of its nature—whether a new growth or a lesion of an ulcerative nature. It is also possible, by the same procedure, to ascertain the existence of intestinal prolapse, or the presence, at or near the brim of the pelvis, of an offending appendix or ovary, of an abscess, or of a malignant tumor springing from bone. Finally, this method of exploration enables us to learn the condition of the bladder, prostate, etc., in the male, and that of the uterus and its appendages in the female. Far too many instances of irremedial cancer of the rectum, vagina, and uterus come to light in consultation practice to warrant the belief that commendable forethought is always practised by the medical attendant first in the field. We have no hesitation in asserting that the loss of life and the great misery caused by these afflictions will be lessened decidedly when an inflexible rule is adopted of examining the rectum, the vagina, and the uterus, when practicable, in all instances in which advice is sought for the relief of ailments of these or of contiguous parts.

Fæcal incontinence owes its origin to localized loss of power of the sphincter muscle, and this in turn may be due to a variety of causes, *e.g.*, overdistention, relaxation, incision, rupture of the perineum, etc. Painful defecation, with or without rectal tenesmus, comes from various causes. Among them may be enumerated: an anal fissure, a fæcal mass, an inflamed prostate, a rectal cancer, and an inflamed or retroflexed uterus. Rectal tenesmus often causes great suffering, and it is especially frequent in those who would unwisely endeavor to gain relief by persistent straining at stool. Impacted fæces, foreign bodies in the rectum, prolapsed hemorrhoids or prolapsed mucous membrane, cancer of the rectum, rectal polypus, dysentery and inflammation of the bowel, and intussusception will cause more or less tenesmus. Rectal tenesmus is not infrequently associated with painful defecation, often adding much suffering and prostration to an already painful infliction. Voluntary resistance to the desire, the adoption of the recumbent posture, and prompt exploration of the bowel to ascertain the presence therein of an exciting cause are the measures which should be taken for the relief of the suffering.

The Stools.—The frequency, shape, color, consistence, odor, and constituents of the stools should always be noted, especially in cases of intra-ab-

dominal injury and disease. The frequency of defecation in disease and in injury should be compared with the habit of the patient in health, due allowance being made for the kind and quantity of food which he eats and for any medicaments which he may have taken.

The size and shape of a stool are regulated by the amount and the consistence of the fæces, the shape and size of the canal, and the degree of the force of expulsion. A small round stool suggests anal prolapse or an annular stricture of the rectum, and it may attend intussusception; the ribbon-shaped stool indicates the presence of large hemorrhoids, an enlarged prostate, or spasm of the anus, and it sometimes attends stricture of the rectum. The color of the stools is modified by the kind of food ingested, by anything that interferes with bile formation and discharge, by medication, etc. Milk and starchy foods predispose to light yellow stools; dark-colored fruits and fluids darken the stools, changing them often to correspond with the substances ingested; interference with the proper discharge or with the formation of bile causes light or clay-colored stools, according to the degree of the interference, and suggests the presence of cholelithiasis, cancer, etc., or structural change in the liver, pancreas, or duodenum. The green stools of infancy and the dark ones of any age indicate, respectively, bacteriologic coloring and the effects of iron, bismuth, etc., medicinally employed. Red and tar-colored stools indicate the presence of blood, the former coming, as a rule, from the lower bowel, especially the rectum. If the stools are red and if they originate from some point much higher up than this, the amount of the blood must, at the time of the hemorrhage, have been large and the expulsion rapid. The tarry stool indicates the occurrence of hemorrhage high up in the digestive tract, or in the upper part of the lower bowel, or in the small intestine, or, perhaps, even in the stomach; or it may indicate slow expulsion of the intestinal contents, and also the influence of the digestive process on the blood that has escaped into the intestinal canal. Microscopical or chemical and spectrum examinations may be needed clearly to establish the presence of blood in a stool. The consistence or density of a stool is often a matter of much surgical significance, as related to the presence of impacted fæces causing diarrhœa, as indicating the degree of intestinal constriction consistent with the passage of a stool of a given diameter, as denoting in a degree the amount of the discharge attending an ulcerative process in the intestine or the escape, into the gut, of the contents of a contiguous abscess or cyst. Scybalous masses passed with or without the aid of enemata are often of important significance as related to intestinal obstruction and to medication. The naturally offensive odor of a stool is increased to near the line of putridity by the diminution or absence of bile in the intestine, as in obstructive disease of the gall ducts, in specific or malignant ulceration of the intestine, and in the gangrenous processes of dysentery. Long-retained, unabsorbed nutritive enemata not infrequently

lead to highly offensive stools. In suspected intestinal obstruction the escape of unusually offensive flatus is often happily followed by a reassuringly copious stool.

A stool in the normal state is composed almost entirely of the products of the digestive tract and the associated organs, and of those portions of the food which cannot be digested. Morbid processes, however, may increase the amount of mucus and even stain it with blood, as in inflammations of the mucous membrane of the intestine and in intussusception. Sloughing mucous membrane may add to the stool membranous shreds; disease of the pancreas due to injury or to a calculous obstruction may add fat; gall stones, too, are sometimes found in it, and they may be of such a size as to cause complete intestinal obstruction. Pus, foreign bodies, and various kinds of parasites also invite attention in special instances.

The Respiratory System.—The careful study of the topography of the thorax, when it is in an active as well as when it is in a quiescent state, a thorough knowledge of the means of making a physical examination of the chest and of the signs elicited by such an examination, along with an understanding of the normal and abnormal rhythm of respiration, constitute the minimum of knowledge requisite for an intelligent appreciation of the common respiratory phenomena of injury and disease.

It is important for the surgeon to note, not only the limits of the pleural cavities in health and in disease, but also the relation of the lungs to the pleura and to the chest walls; otherwise he may imperil the patient's chances by a needless invasion of a pleural cavity or of the lungs. Since these relations are much changed by the deep respiratory act, the importance of making an incision into the pleural cavity midway during an inspiration or an expiration ought to be apparent. That the pleura in health extends in the mammillary, the midaxillary, and scapular lines about two, three and one-third, and one and one-half inches, respectively, lower than the corresponding borders of the lungs is a matter of much practical significance in thoracic operations and in abdominal operations related to the lower border of the ribs (see remarks on p. 523 with regard to the area of liver dulness). The important points in chest topography, so far as it relates to diagnosis, are the following: the clavicle corresponds to the first rib; the projection at the junction of the first and second pieces of the sternum corresponds to the second rib; the nipple, in the male, corresponds to the fourth intercostal space; a line passed over the nipple around the chest crosses the sixth interspace at the axillary line (important in tapping the chest); the cartilage of the seventh rib and the ensiform process form an epigastric angle; the cartilage of the ninth rib corresponds to the gall bladder; and the eleventh and twelfth ribs can be located outside the erector spinæ in stout persons.

Inspection.—The inspection of the chest in health and in disease, if intelligently

conducted, is of great significance. Ordinary inspection may be supplemented with advantage by the use of the x-ray. One should note the frequency, the type, the character, and the rhythm of the respiratory acts. It is also important to observe whether or not the normal costal breathing is modified by the presence of disease or injury of the pleura, the lungs, or the diaphragm, or by disease of the abdomen from any cause; whether or not the diaphragmatic type is changed from the normal by pleurisy, pleurodynia, intercostal neuralgia, peritonitis, paralysis or spasm of the abdominal muscles, fracture of a rib, etc. Nearly all forms of disease increase the frequency of the respiratory act. Narcotic poisoning and disease or traumatism of the respiratory centre lessen the frequency of respiration. Cerebral compression changes the character of respiration, which becomes stertorous.

Significance of the Changes in the Character and Rhythm of the Respiratory Acts.—Increased inspiratory effort points to an obstruction to the entrance of air, dependent on an impediment in the larynx or trachea; œdema of the glottis and pressure from an aneurism upon the trachea are the two pathological conditions which ought to be thought of in this connection. This kind of obstructed inspiration is attended by increased expansion of the subclavicular regions and by retraction of the supraclavicular and intercostal spaces, and of the epigastric area. Labored expiratory effort is attended with bulging of the intercostal spaces; emphysema and asthma being the common causes. In the former disease bulging of the soft parts above the clavicle may be observed during inspiration. The modifications in rhythm include the Cheyne-Stokes respiration and the sensory and pupillary phenomena so often associated therewith. This form of respiration is noted especially in grave cardiac, renal, and cerebral disease and as a result of certain injuries; and it is sometimes observed in the typhoid and septic states associated with pneumonia and the eruptive fevers. The jerking respiration of acute pain in the chest, as in fracture of a rib, the snoring breathing of the coma of disease and of narcotic poisoning, and the noisy respiration of faucial obstruction or paralysis are familiar phenomena and aptly illustrate modifications in respiratory rhythm.

The uniformity of expansion of the chest attendant on normal respiration is modified in a striking degree by disease and by excitement. Dyspnœa dependent upon some cause or other may be associated with a chest which remains of the same degree of expansion during both inspiration and expiration. Mental excitement and physical effort are each capable of producing the same effect. Usually, however, such a condition of things suggests the existence of actual disease, as, for example: some lesion that interferes with the proper entrance of air into the lungs; a loss of respiratory power; a traumatism of such a nature that the patient voluntarily fixes his chest in order thereby to escape pain; acute disease involving both sides of the chest. Deviations confined to one

side of the chest usually imply disease or injury of its bony framework, and the lack of mobility of the affected side is commonly emphasized by the greater mobility of the sound one, which is performing compensatory work. Deformity of the thorax may be partial or general in extent, of transient or permanent tenure, of trivial or serious aspect, and easy or impossible of correction.

Various instruments are employed for determining the dimensions, the respiratory capacity, the mobility, and the surface outlines of the chest. Information with regard to these, however, cannot properly be given here; the reader must seek for it in the special articles.

In a general survey of a patient's chest it is desirable to note carefully the condition of the surface circulation. In a normal state the superficial veins are not especially noticeable, but they may become so enlarged, through the influence of some disease which interferes with the circulation within the thorax, that their tortuous outlines are plainly visible. In a case of long standing, which recently came under my observation, the patient's face, ears, eyes, tongue, and throat were so engorged as to give him, for a time, until the obstructed circulation had regained its equilibrium, a distorted mien. It was found that the obstruction existed in the interior vena cava at a point located just above the heart. As a result of this obstruction the capillaries and veins corresponding to the attachment of the diaphragm to the thorax were much enlarged, and became rapidly and enormously distended because of a temporary increase in the obstruction; and as a further result there was venous engorgement of the structures of the body which empty their blood into the channels of return circulation above the diaphragm.

It will not be amiss at this time to direct attention to the following anatomical facts which have an important bearing upon the question of diagnosis:

Litten's Diaphragm Phenomenon.—This manifestation of diaphragm action occurs in thin persons, and is best seen by placing the patient on the back in a good light and inspecting the chest at an angle of forty-five degrees. It consists of a shadowy line which lies at an acute angle to the ribs and travels downward a distance not exceeding two and one-half inches (in forced inspiration) in harmony with the downward movement of the diaphragm. The manifestation is seen to begin at the sixth interspace on both sides with inspiration, and to move downward to the free borders of the ribs as the inspiratory act is completed. In expiration the reverse of the movement is noted. The vital capacity of the lungs is thought to be proportionate to the width of the shadow. Eliot has called attention to the increased depth and the fixity of the costal arch in the presence of disease of the contiguous viscera. Normally, the arch is mobile and quite easily encroached upon by pressure against the lower ribs. In disease, however, it is wide and fixed. Harrison has described a groove that appears on the chest of rickety subjects and is associated with impeded inspiration due to obstruction of the nose, fauces, and bronchial tubes, from various

causes. This grooved line begins at the xiphoid cartilage, and extends along the arched surface of the thorax to the axilla on either side.

The Circulatory System.—*The Heart.*—The established position of the heart in the chest in health and the ordinary phenomena attending its activities are matters of such common knowledge as fortunately to require no special mention here. The apex in health in the adult is noted in the fifth intercostal space, just inside the mammillary line. Prior to the tenth or twelfth year the apex beat is in the fourth intercostal space, at or just outside the mammillary line. In making these observations it is essential to note if the nipple is displaced from the normal site; also to remember that changes of position in health are attended with change in the site of the apex beat. If the body be inclined to the left, the apex beat approximates the midaxillary line; with a full inspiration it is moved downward and to the right. In old age the apex beat is depressed, and in transposed viscera the change in the site of the beat is correspondingly modified. Deformities of the chest incident to spinal disease or to some other cause change the relations of the heart-beat to the chest wall. Diseases of neighboring viscera, characterized by the pushing influence of fluids, gases, solid and aneurismal tumors, etc., and by the pulling effects of contracting organs and adhesions, aided or not by abdominal distention, often change the position of the entire organ to a degree comparable with the extent and activity of the morbid process. Hydrothorax, pneumothorax, tumors and phthisis of the pulmonary tissue, aneurism of the aorta or of the contiguous vessels, and abdominal distention, encroaching on the diaphragm, are some of the pathological conditions which may produce a displacement of the heart. Hypertrophy, dilatation, and aneurism of this organ, and pericardial effusion increase and change the area of cardiac dulness in accordance with the degree and direction of the modifying process. In children with rickets or with marked hypertrophy or dilatation of the heart, or with pericardial effusion or aneurism of the heart, the precordial region may be bulging.

Thrills and friction sounds are appreciated by the hand, the tremors corresponding to the sites of the valves of the heart and the direction of the blood flow. Aneurism of this organ also gives to the hand of the examiner the characteristic thrill. The friction sound of pericarditis, traumatic or otherwise, is often readily distinguished by means of palpation; auscultation announces the absence or feebleness of heart sounds, their location, rhythm, the seat of greatest intensity, also the presence of abnormal action, enabling one to judge of the need of treatment and of the value of the remedial measures employed, especially on urgent occasions.

The heart sounds are intensified by a variety of causes, *e.g.:* approximation of the organ to the ear, as may be effected by pressure from the outside; thinness of the thoracic wall; hypertrophy and increased action of the heart; upward pressure upon it by tumors; pericardial adhesions, etc. The heart sounds

are diminished by conditions diverse from those just preceding, such as, *e.g.*, a thick chest wall, distention of the lung or of the pericardium with blood, pus, or air, and weakened heart action from any cause. The splashing sound heard on auscultation indicates the presence of air and fluid in the pericardium. All of the foregoing are of importance in cases of traumatism of the heart. The significance of the various murmurs, etc., may best be considered in books and articles devoted to the purpose. The duskiness of the surface of the skin attending general obstruction of the circulation, and the paleness from feeble or depleted blood supply, need only be mentioned to establish their significance.

The Vessels.—The pulsation and enlargement of the large arteries of the extremities and neck are a part of the history of old age, cardiac hypertrophy, excessive physical exercise, excitement, etc. These phenomena, when observed in the neck, signify aneurism of the aorta, atheroma, Graves' disease, etc. Undue enlargement of the carotids and of the innominate, in such cases, is sometimes mistaken for aneurism. Pulsation of the abdominal aorta in thin, nervous, or apprehensive subjects is frequently the cause of much distress, and may be mistaken for aneurism, especially when associated with pain, enlarged pancreas, or a tumor of the stomach, omnetum, or colon. The epigastric pulsation which is often observed in these cases, or which may be transmitted from above by a hypertrophied heart directly, or by contact with the liver, is also often a source of needless solicitude to the patient and possibly to the medical attendant. However, in all these cases a careful differentiation of the phenomena observed from those which attend a case of genuine aneurism ought soon to clear up all doubts. And in this connection it is well to examine the smaller arteries, with the idea of noting their shape, size, and pulsation as related to arterio-sclerosis.

Of much significance are the pathological changes to which the veins are liable. A sudden pain in the calf of the leg or in the instep, the tenderness and hardness in the course of the femoral or iliac vein, followed by swelling of the leg and foot, are so commonly an important part of the history of crural phlebitis as to require no more than mention at this time, unless it be to warn against the possibility of mistaking it for rheumatism, as is frequently done. In ligaturing a distended vein, in a case of varicose veins of the lower extremity, it has sometimes happened that the ligature has included a contiguous important nerve, thus greatly aggravating the original pain due to the distention of the vessels. The surgeon, therefore, needs to be on his guard against this accident. When the veins of the arm or the leg become gorged with blood and the skin of the affected limb takes on a dusky hue; and when, furthermore, these changes are soon followed by the development of œdema and pain in the parts involved, we may unhesitatingly assume either that a thrombosis has occurred in the chief vein or that it is being pressed upon by some pathological product at a point in or not far distant from the axilla (in the case of the

arm) or the groin (in the case of the leg). For example, a cancerous growth in the axilla or in the pelvis is a common cause of such a sequence of events. In this connection it should be said that the pressure of a tumor of the mediastinum or lung, or of an aneurism of the arch of the aorta, or of the subclavian, the axillary, or the innominate artery (right side), or pressure from enlarged axillary lymph nodes, may each be followed by discoloration and œdema of the corresponding extremity. It must not be forgotten, however, that the free clearing out of the contents of the axilla for cancer may be followed by painful and excessive œdema, because of the resulting destruction of lymphatics —changes which may be confused with œdema from pressure of lymph-node involvement attendant on a relapse of a malignant growth. Enlargements of lymph nodes and morbid growths pressing on the iliac veins of either side cause œdema and pain of a lower extremity. Finally, there remains the possibility that the œdema may be due to other and general causes.

The enlargement of the superficial abdominal veins, taken in connection with the fact that the blood in them courses in the reverse direction, indicates the existence of some obstruction to the portal circulation. (See also the instance of obstructed circulation in the superior vena cava referred to on p. 529.) The veins of the mucous surfaces are not infrequently dilated, because of portal obstruction and of the hindrance of the circulation incident to arteriosclerosis. The hemorrhoidal and the spermatic veins and those of the broad ligament, etc., sustain special inflictions of a varicose nature, which may be mistaken for disease or other morbid processes of these regions. The veins of the neck are large and quite superficial, and the varying conditions serve as an important index of many conditions. For example, a collapsed jugular indicates thrombosis of the lateral sinus, especially when pressure on the vein above the clavicle is not followed by distention of the vein. Distention of the jugulars attends coughing and straining efforts in health. Obstructed pulmonary circulation and obstruction to venous return from pressure on the innominate and other deep veins of the neck and chest, as from tumors and aneurism, cause free engorgement of the veins of the neck. The indistinct influence of an inspiratory effort on the jugulars in health is rendered more distinct when these vessels are engorged from any morbid cause. Pulsation of the jugulars may be transmitted from the contiguous carotids, or it may be due to obstruction of the return flow, as in the case of an intrathoracic tumor or aneurism, in mitral regurgitation, in interference with the pulmonary circulation from any decided cause, and in increased intrarenal pressure. The valve at the bulb (junction of subclavian vein with the jugular) commonly arrests the upward flow, but in long-standing or decided obstruction dilatation of the vein follows, and this impairs or destroys the usefulness of the valve in preventing a return flow. Murmurs are sometimes heard in veins; they are usually of anæmic origin.

The pulse is interrogated to determine the force, frequency, and rhythm of the action of the heart and the degree of tension of the arteries. The pulse should, in every instance, be taken under conditions which are, as nearly as possible, alike; and, when it is practicable to do so, the test should be made at established intervals. A change in position of the patient, excitement of any kind, active digestion, the time of day, etc., are each of important moment in estimating the proper significance of the heart's action and the state of the pulse. A full minute of estimate of the pulse is better than a fractional estimate multiplied to equal the count of a minute. The latter practice is apt to increase the numerical record in proportion to the smallness of the fraction of the minute, especially when a full measure of the fractional count is the multiplicand.

In taking the pulse it is well to test the radial arteries of both sides, especially when the patient shows signs of prostration; for an unusually small vessel of one side may mislead the surgeon in properly estimating the patient's condition. The temporal, femoral, or carotid vessels may be selected for estimating the pulse when for any reason the radial at the wrist is not available, but at the same time it must be remembered that the nearer to the heart and the larger the vessel the more pronounced is the action. The slow and feeble pulse of cerebral compression, the rapid and feeble one of cerebral and other kinds of shock, and the diminished frequency (bradycardia) of pulsation in increased arterial resistance, in arterial sclerosis of the medulla, in Bright's disease, in bile poisoning, in aneurism of the heart, in convalescence from acute disease and depressing injury, etc., are not infrequent exhibits of the heart's action under these varying conditions. The increased rate (tachycardia) of pulsation in Graves' disease, in the abuse of the use of narcotics, in excessive sexual indulgence, in neurotic states, in loss of blood, etc., is of significant import. The pulse beat in children is naturally and markedly of greater frequency than in adults.

Blood pressure is a matter of importance and can be sufficiently well estimated by one who is experienced in the taking of the pulse. The information gained in this way can be promptly utilized in estimating the degree of shock or the amount of loss of blood in a particular instance. Ordinary experience determines the presence of the hard, wiry pulse of acute peritonitis. However, the employment of special instruments for the purpose of scientifically recording the blood pressure is much the better and more reliable plan. The various examples of sphygmographs and sphygmomanometers are to be found in the article on Blood Pressure in Surgical Conditions.

The Temperature.—The technique of taking the temperature is of such common knowledge as to call for no mention here. A well-tested and reliable thermometer should be employed at all times. Whether or not the mouth, the axilla, the rectum, or the vagina be selected as the proper site will depend

on the circumstances of the case. A sense of delicacy may render it inadvisable to use for this purpose the vagina or the rectum; a sense of expediency may make us hesitate about using the mouth, especially in the imaginative, the young, and those not in proper control; but the demands of accuracy will approve of the selection of whichever place will furnish us with the most trustworthy results. In any event it will readily be admitted that it is always wise to cause the instrument to be cleansed in the presence of the patient before using. Care should be exercised in all instances in taking the temperature and in recording the results. When efforts at deception are suspected, only vigilant and protracted observation, amounting to keen surveillance, will defeat the purpose of those who are interested in the endeavor to deceive. In estimating the importance of the temperature record in disease it is well to recall that the normal temperature of the rectum and vagina is higher than that of the mouth, and is less liable to accidental variations; on the other hand, the temperature of the axilla is almost a degree lower than that of either of the mucous channels already mentioned, and only with comparatively great care can a correct record be made in this locality. Normally, the temperature of the body is highest during the daytime, and is lowest at from two to four in the morning—facts which are to be considered in estimating the fluctuation of the temperature in disease at the time mentioned. The frequency of taking the temperature is a matter both of custom and of demand. The intervals of custom are a matter of choice—once in three or four hours, when practicable. When the circumstances of a case demand the adoption of a certain interval we must conform to the needs of the case. In any instance neither over-anxiety nor exceeding diligence should be permitted to measure the interval. Physiologically, youth, exercise, digestion, excitement, and heated environment increase the temperature. Old age, cold, and depressing influences lower it. The normal temperature is 98.6° F.; but shock, loss of blood, starvation, wasting disease, cerebral abscess (often), myxœdema, and great prostration are attended with a subnormal temperature (97° F.). Sometimes a sudden drop to 94° F. or less attends the intermittent manifestations of pyæmia, with abscess complications, and of the liver infections. High temperatures of such romantic altitudes as 150° F., more or less, need not be considered seriously in connection with the morbid processes of physical disease. One of 122° F. appears to be entitled to respectful consideration. Personally, I have not yet observed a temperature above 110° F.—once in a case of hysteria (probably deceptive); several times in cervical injury involving the spinal cord, soon followed by death of the patients; and once in insolation, also followed by death.

The giving of the coal-tar products in advance of a diagnosis of the cause of the fever, for the purpose of reducing the temperature, is an error often committed, and always invests the case with an element of needless uncertainty that should be avoided.

The relation of a patient's body temperature to the time of operation is a matter of decided significance. A fever (100° to 103° F.) developing within the first twenty-four hours after an operation, attended with no obvious symptoms, and disappearing within three or four days, is characterized as an "aseptic fever" or as "post-operative fever," and is due to the presence in the blood of irritating products (nucleins, etc.) resulting from the injury. When, however, the body temperature remains elevated for three or four days, and other symptoms appear, or if, after becoming normal, the temperature again rises, the wound should be examined at once, as infection is quite certain to be present. When, two or three days after an operation, chilly sensations and discomfort, followed by a rapidly rising temperature (102° to 104° F.), occur, with thirst, headache, pain in the wound, and tenderness and swelling of the adjacent soft parts, genuine *surgical fever,* due to the absorption of the toxic products of fermentation bacteria, is at hand. When the temperature rises to a considerable degree (103° to 104° F.), with morning remissions and evening exacerbations and a well-defined chill; and when, at the same time, the wound appears dusky and œdematous and is quite tender, *suppuration fever,* due to the absorption of the toxins of poygenic organisms, is present. The temperatures of sapræmia, of septic infections, and of pyæmia are quite characteristic in their curves. (See the article on this subject in the present volume.) A high temperature with or without a chill in the presence of an operation wound of quite normal appearance, suggests the onset of erysipelas; and, if the suggestion prove true, there will soon be seen unmistakable local evidences of the disease. The existence of a temperature suggestive of the presence of pus, with no evidence at the seat of the injury corroborating such suspicion, often demands a scrutiny of the most searching character to reveal the seat of the trouble.

The Nervous System.—For the purpose of this article it will be sufficient if we devote our attention to sensation, motion, reflex action, the modifications of special senses, and the changes in nutrition of various parts. The senses of touch, of pain, of heat, of locality, of pressure, etc., are of varied importance in their relation to diagnosis. *The sense of touch* may be exaggerated (hyperæsthesia) in neuralgia, neuritis, and disease of the spinal cord, and it forms a part of the history of hysteria and spinal irritation. It may also be exaggerated by the effect of local irritants. The sense of touch is diminished (hypæsthesia) in disease and injury of the posterior columns of the cord, of the posterior part of the internal capsule, of the parietal lobe, and of the pons. The insane, the defective, and nervous subjects may suffer in this manner. A total loss of the sense of touch (anæsthesia) follows destructive lesions of a special nerve, of the spinal cord, or of the brain, when the lesion destroys the related functional continuity in either of these structures. If a nerve be destroyed the effect is local; if the cord be at fault, sectional anæsthesia of the body is present when the damage involves both sides, and anæsthesia is noted

on the opposite side of the body when only one side of the cord is injured. In a lesion of the brain anæsthesia is commonly associated with hemiplegia when the latter is present. In a circumscribed lesion of the cortex only an extremity may suffer anæsthesia. In functional anæsthesia of one-half of the body, areas of irregular or of symmetrical outline are present.

The sense of pain (algesia) differs in different races and in various persons. The Teutonic and Slavonic peoples suffer less, it is thought, than do others It should be appreciated by all concerned that persons of dull perceptions and phlegmatic natures are less sensitive to pain than are those of an opposite character. Habitual hardship, religious and other kinds of excitement blunt the sense of pain; but refined restraint and associations and long suffering unfit the sensibilities to bear pain. The surgeon should be able after a little time to differentiate between those who bear severe pain uncomplainingly and those who for different reasons magnify their sufferings. In any instance indifference, heedlessness, or superficial examination on the part of the surgeon may be followed by discomfiture, criticism, and loss of professional station. The kinds of pain are numerous, and to each kind can often be attached a special degree of significance, particularly as indicating the seat of disease and the variety of tissue involved. Acute pain of a distressing, lancinating character is a sure accompaniment of an acute inflammation of a serous (especially) or synovial membrane. The pains of neuritis, and of neuralgia, and those caused by a tumor and by aneurismal pressure are often severe and radiating. Dull pain occurs in acute inflammation of viscera, in chronic inflammations generally, and in connective tissue, in which latter case a throbbing sensation is frequently present when this tissue is acutely inflamed. Itching of often tormenting nature characterizes acute inflammation of mucous surfaces, especially in conjunctivitis, pharyngitis, and urethritis. Burning pain marks inflammation of the skin, as in erysipelas, sunburn, and in other local irritating reactions. Boring or grinding pain is indicative of disease of bone or of periosteum, of the pressure of an aneurism on bone, and of gastric ulcers. Sickening pain characterizes acute disease of, and especially traumatic pressure on, the testis. Throbbing pain happens especially with boils, carbuncles, plantar and palmar abscess, and in whitlow, and is dependent on the confinement of the morbid process by unyielding overlying tissues. Paroxysmal pain and shifting pain are characteristic of such diseases as neuralgia, colic, rheumatism, and locomotor ataxia.

Ordinarily, the location of the pain corresponds to the seat of the morbid process causing it. Sometimes an injury done to the main trunk of a nerve, or to one of its branches, will give rise to pain, not at the seat of injury, but at the area supplied by such nerve. This is known as transferred pain. Such a transferred pain occurs in cases in which the trunk of a nerve or one of its branches is pressed upon by a tumor or is subjected to some other form of irritation.

Then, again, in amputation for an irritable stump or for disease or traumatic destruction of a foot or leg, it often happens that the distress continues after the operation and is assigned by the patient to the amputated part. The pathologic change which takes place in the end of the nerve, in a stump, or in a nerve which has been included in a ligature, produces a sensation like that caused by the original infliction. Pain at the inner side of the knee in disease of the hip, in the testis in renal disease, in the nipple in uterine disease, in the dorsal region in disease of the stomach, in the sacral region in disease of the uterus, in the abdominal wall in Pott's disease of the spine and in some cases of pneumonia or pleurisy, especially in children—these are, all of them, examples of transferred pain, sometimes called reflex.

The determination of the differences in the pain sense (algesia) in different persons, in the different parts of the body, and in different diseases, is effected by various devices and by the hand of the diagnostician. The manual method is a good one when the temperature of the parts and the pressure exercised are practically adjusted. In the use of any measure for this purpose care should be exercised not to confuse the records of the results. A more extended statement of these matters can be found in books devoted to special diagnosis. Hypersensitiveness to pain (hyperalgesia) of the keenest nature sometimes appears in inflammation, and in nearly all instances *tenderness* is a part of the local history of inflammation and of many other disease processes. The opposite of these conditions (hypalgesia) betokens a lesion of the nerve, of the spinal centres, or of the focal area of the brain. The integuments of idiots and epileptics, and also of parts of the body continuously exposed to irritating contact, present this manifestation. A loss of the sense of pain (analgesia) is a specially important manifestation, indicating destruction of nerve tissue from injury, transverse spinal myelitis, a tumor, or an injury of the cord, disease of the posterior part of the internal capsule, or disease or injury of the parietal lobe. Insanity, hysteria, and hypnotic suggestion may take a part in the causative history of this change, and in the last two instances the exhibit is often of irregular, singular outline. Syphilis may cause analgesia.

The Heat Sense (thermo-æsthesia).— The heat sense enables one to recognize the differences in the temperature of various things and of different surfaces. The sense of heat may be in abeyance in a part, independently of that of cold, or the reverse may happen; also these senses are sometimes confused with each other. A complete loss of the sense of heat (thermo-anæsthesia) occurs in instances of destructive nerve lesions similar to those found in analgesia, and consequently is a symptom of great importance. In pressure myelitis and in involvement of the gray matter of the cord exclusively, tactile sense is retained, but the temperature and pain senses are lost (Musser). A loss of tactile sense, with the loss of pain sense, happens in injury of the trunk of a peripheral nerve.

The sense of locality varies in different parts of the body, being most evident

on the lips and least evident on the body between the scapulæ. This sensation is lessened in the various forms of hyperæsthesia, especially of central origin, in tabes dorsalis, and in injuries of the parietal lobe. The full significance of the pressure sense has not yet been decided, but that it is of importance in indicating the structural changes incident to ataxia and paralysis is evident.

Impairment of the muscular, articular, and tendinous senses occurs in cerebral ataxia, cortical lesions, and those of the crura and pons; also in transverse injury or disease of the spinal cord.

Motion.—The manner of standing and that of moving are fruitful sources of inquiry in the affected and in the well. In the latter, they are important as indicating the type of the individual; in the former, the apparent deviation from the normal indicates disease or injury. A normal person, standing with the feet close together and with the eyes open, will sway forward and back and from side to side about an inch in each direction. The loss of the muscular, articular, and tendinous sensations, as in locomotor ataxia, greatly increases the swaying, and the patient is likely to fall if the eyes are closed. Disease of the middle cerebellar lobe and aural vertigo likewise cause this ataxic state. In other respects the manner of standing is suggestive of the infirmity of disease or of old age. The bending forward, as in paralysis agitans, in spinal disease, in old age, and in some instances of intoxication; and the bending backward, as in pregnancy or in increased abdominal weight from any cause, and also in instances of intoxication, are manifestations of significance. Whether or not the limbs be alike or be specially distorted are circumstances which should be noted.

As in standing, so in walking, the body is bent backward with increased weight in front, and the feet are more widely placed to insure a firmer support. That limping and halting gaits distinguish the impairments of rheumatism, disease of joints, etc., are facts which require no special mention here. The ataxic gait of locomotor ataxia, the gait of alcoholic intoxication, of cerebellar tumors and of cerebellar ataxia, the "prancing" gait of paralyzed flexors of the foot, the spastic gait (rigid and stiff-moving limbs and shuffling step) of lateral spinal sclerosis, the involuntary hastening gait of paralysis agitans, the waddling gait of lordosis, of congenital dislocation of the hip, and of pseudo-hypertrophic muscular atrophy, are each not infrequently seen.

Reflex Action.— Three kinds of reflexes will be mentioned—the cutaneous or superficial, the tendinous or deep, and certain organic special reflexes. Reflex action involves the passing of the peripheral stimulus along an afferent (sensory) nerve to the motor cells of a nerve centre in the spinal cord or medulla, and the changing of the stimulus by these motor cells into a motor impulse, which is reflected along an efferent nerve (motor) to a muscle which consequently involuntarily contracts. It therefore follows that these three nerve factors and the muscular factor necessary in a reflex action should each be

healthy if a proper response is to be obtained, and that when either factor is out of order the reflex result is correspondingly affected. The irritation of the skin at the selected site by stroking, picking, pinching, etc., by heat or cold, or by chemical irritants, and perhaps by a breath or a breeze, will in the normal state cause the desired result. Generally, conditions increasing muscular tone increase the reflexes, and opposite conditions lessen them. Reflexes are lessened when the attention of the patient is engrossed in the procedure, and increased if the patient be required to make a severe muscular effort, such as the clenching together of the fingers of each hand. In the coma of uræmia and of saccharine diabetes, attended with lessened muscle tone, exaggerated reflexes sometimes appear, especially when the muscles are much relaxed (Musser).

The common superficial reflexes are the scapular, the epigastric, the abdominal, the cremasteric, the gluteal, and the plantar, belonging to the spinal cord; and the conjunctival, the pupillary, and the palatal, which are connected with the medulla. The palatal reflex is lost in bulbar paralysis and in hysteria. The remainder of these reflexes are interesting, and each in turn signifies the state of the nerve centre presiding over it.

The Deep Reflexes.—The deep reflexes are more particularly those of the knee and ankle, to which may be added special ones of the foot. The reflexes of the jaw, the elbow, and the wrist, although not as constant as are the preceding, are frequently sought for, and when present they are given the proper diagnostic significance.

The knee-jerk, or patellar reflex, is invariably present in health. The absence of this reflex, therefore, signifies disease or injury of one or more of the factors of the reflex arc. Hence the loss of the jerk in neuritis, in disease of the posterior roots and columns (locomotor ataxia), in disease of the anterior horns (poliomyelitis), and in transverse myelitis of the second and third lumbar segments. The shock of cerebral hemorrhage, traumatic compression of the brain, and traumatism of the spinal cord may cause abeyance of the jerk; and it may be wanting in diphtheria and in other diseases of decided toxic nature. Exaggeration of the movement indicates the presence of a competent reflex arc minus the inhibiting influence of cerebral cells or of their transmitting fibres in the lateral pyramidal columns. Increased irritability of the spinal cord exercises a similar influence. In apoplectic hemiplegia (shortly after), in cerebellar ataxia, in sclerosis of a lateral column, in transverse myelitis and injury, in the pressure exerted by a tumor, and in unilateral lesions of the cord above the reflex lumbar centres exaggerated movement takes place. In a unilateral lesion the increase of movement is on the affected side. In tetanus, strychnia poisoning, and hysteria, and in spinal irritation, this manifestation is present.

Ankle clonus has the same significance as the knee-jerk phenomenon. Tapping of the hamstring tendons or the inner condyle of the tibia causes adductor

reflexes, which, however, are not of independent clinical significance. Ankle clonus or tendo-Achillis reflex is present in nearly all healthy persons. Ankle clonus is present in organic disease and may be noted in functional trouble, and even then organic change should be suspected until disproved. The presence of exaggerated ankle clonus or ankle-jerk in a case is symptomatically equivalent to the presence of exaggerated knee-jerk. Lesions of the motor regions of the brain, transverse myelitis, lateral sclerosis are causative of ankle clonus. If the great toe be flexed on the sole, the foot on the leg, the leg on the thigh, and the thigh on the body, and if at the same time the great toe be tapped on the tendon, Sinkler's reflex will appear. If, with the lower limb extended, the inner surface of the sole of the foot be stroked with the hand from the heel upward, the toes will flex in health; but if, instead, the great toe be extended, either alone or with the others, Babinski's reflex is produced, indicating in both instances transverse injury of the spinal cord, as may happen in fracture of the spine, as well as in transverse myelitis, or in pyramidal injury or disease.

The Special Senses.—The special sense of touch has been already considered. The senses of sight, smell, taste, and hearing will be very briefly mentioned.

The Sense of Sight.—The palpebral fissure and the pupil are the openings through which light reaches the eye. The unopposed raising of the lid in unconsciousness and the twitching opposition attended with upturning of the eyeball in hysteria are significant features. The swelling of the lids and protrusion of the eyeball incident to cerebral thrombosis, and the prompt protrusion of the ball and extravasation of blood beneath the upper conjunctival fold in fracture through the anterior fossa of the skull, are important manifestations. Contusion of the supraorbital ridge or of the contiguous area, attended with rupture of vessels, may cause an extravasation of blood to develop gradually in the upper lid. Orbital emphysema often is present in fracture of the nasal bones, especially when shortly after the accident the patient makes an effort to expel the nasal contents. The deviation of the eyeball from its normal position should be observed, and its relation to injury at the base of the brain, to tumor development, to intracranial disease, and to functional change should be carefully noted, and estimated by consulting special sources of information.

The Pupil.—Before examining the pupil, the surgeon should pay careful heed to the precautionary requirements commonly stated in books and so essential to the securing of intelligent findings. A changed outline of the pupillary margin suggests the activity of syphilis, of rheumatism or gout, and perhaps of tuberculosis.

Irritative dilatation of the pupil arises from congestion of the cervical portion of the cord or from meningeal inflammation or new growths in this

region; from high intracranial pressure dependent upon surgical traumatism, cerebral tumors, or other pathologic changes; also from spinal and intestinal irritation and from mental disorders.

Paralytic dilatation of the pupil is present in disease or injury situated at the base of the brain and affecting the nucleus of the third nerve; in sinus thrombosis (late); in cerebral softening; in fracture or dislocation of the cervical vertebræ, with injury of the cord (cilio-spinal centre). Involvement of the cervical sympathetic by a tumor or an aneurism in the neck may cause the myosis indicative of a paralyzed sympathetic or the mydriasis of an irritated one. Fractures of the cervical spine involving the cord will for similar reasons produce a like effect on the pupil of one or both sides.

Irritative contraction of the pupil happens in meningitis, in traumatism, in intracranial pressure on the third nerve or its nucleus, in hemorrhage, in tumor, in abscess, and also in disorders due to other causes. This form of contraction follows excess in the use of tobacco and overstrain of the eyes. Traumatism or pressure involving the cervical sympathetic may cause contraction or dilatation of the pupil, depending on the severity of the injury.

Paralytic myosis arises from involvement of the cervical sympathetic in fractures or dislocations of the cervical spine, and also from the pressure exerted by a tumor or an aneurism. Degeneration of the posterior columns of the spinal cord and lumbar paralysis, etc., cause it.

The influence of various drugs on the changes in the pupil should not be overlooked in this connection.

It may be useful to remark that the pupil may be contracted in the rapid breathing of Cheyne-Stokes respiration, and dilated in the interval of arrest in the acts. A pupil irresponsive to light and darkness, but responsive to accommodation (Argyll-Robertson pupil), is highly indicative of locomotor ataxia. The pupil dilates in health if the skin of the neck is pinched, unless the sympathetic be destroyed or the patient have paresis or structural change of the eye itself. The alteration in the area and outline of the visual field is exceedingly important in determining the presence of disease and its localization in the brain, and of disease of the visual fields themselves. The wide scope, the great importance, and the special technique of the examination of these cases render it necessary that this work should be done by an expert. Injury or disease of the base of the brain, so located as to involve either the optic nerves or the optic tracts, the chiasm, the posterior part of the optic thalamus, the external geniculate body, the anterior quadrate body, or the visual centre of the occipital lobe, disturbs or destroys sight, according to the degree and extent of the disease or injury.

The Sense of Smell.—The sense of smell plays an important part in surgical diagnosis, giving warning of impending disasters and of the presence of unsavory things. It warns us of approaching gangrene of exposed and pulmonary tissues,

determines the first evidence of feculent vomiting, detects the ammoniacal odors of urinary incontinence, notes the sweet breath of established pyæmia, and otherwise enables one to detect hidden offensive processes, and in a general way indicates cleanliness of the patient, of the fabrics surrounding him, and of the room of his confinement. A modification or loss of the sense of smell may depend on modifying changes in the mucous membrane concerned, on an injury of the olfactory bulb or tract or the uncinate gyrus. A modification in degree or character, or the loss of the sense of smell, may follow a severe blow on the head or fracture of the base, or attend hysteria or result from structural changes in the olfactory centres of the brain.

The Sense of Taste.—This sense, like that of smell, is modified by the state of the mucous membrane related to the function; therefore the condition of the surface of the tongue and of the soft palate is of great importance; also disease affecting the glosso-pharyngeal, the trifacial, and facial nerves is of much significance in this relation. The sense of taste may be lost or modified by basilar meningitis, by a tumor or an abscess at the base of the brain, and by a fracture of the base of the skull involving the facial nerve. Abnormal taste impression occurs from taking bromides and other medicinal agents. Hysteria modifies the sense of taste. The practically intimate association between the senses of taste and smell should be considered, in order that we may avoid the error of supposing that there is a genuine loss of taste, when as a matter of fact this seeming loss of taste is due to some cause which is interfering with the appreciation of odors by the sense of smell.

The Sense of Hearing.—To hear and not to understand the meaning of words is no more embarrassing and not nearly so distressing at times as it is to be unable to interpret correctly the utterances of disease. Therefore an educated ear is of superlative importance in promptly detecting and correctly interpreting the significance of morbid sounds in a patient. The surgeon should be quite as able as the physician to discover by auscultation and percussion evidences of disease of the pleura, the lungs, and the heart; otherwise he is ill prepared to judge of their influence on patients subjected to surgical interference or on those suffering from traumatic injury, and to estimate rightly the effect of anæsthesia in these circumstances. This sense, the same as the preceding senses, is impaired by the state of the external ear, *i.e.*, by the presence of wax, blood, pus, or of foreign bodies in the external auditory canal and by its occlusion through swelling of its walls. Lesions of the auditory nerves, of the posterior part of the quadrate, of the internal geniculate bodies, and of the cortex of the first and second convolutions of the temporal lobe, compromise or destroy the sense of hearing. Extensive disease of the middle and internal divisions of the internal ear, fracture of the base of the skull, contusion and laceration of the base of the brain, tumors, hemorrhage, and inflammation located in this vicinity effect the same result. Syphilis is a fertile

source of deafness. Tinnitus of differing kinds, from established and from indefinable causes, is frequently present. The variety synchronous with the action of the heart and arrested by compression of the carotid is often dependent on vasomotor paralysis, aneurism in the temporal bone or at the base of the brain, etc., and on inflammation of the middle ear. Finally, the importance of morbid auditory sounds can be properly estimated only by the history of the case and by their relations with morbid processes of the brain.

The Reaction of Degeneration.—A comparison between the normal responses of nervous and muscular energy to galvanic and faradic currents and those obtained in disease, is of great importance, indicating, as it often does, the location of disease in the respective factors of the reflex arc, the nature of the process, and the probable outcóme of the affliction. Generally speaking, absence of response to the faradic stimulus, and equal or greater response at the positive than at the negative pole, characterize degenerative reaction. For the means and the methods employed in the securing of these results and in interpreting their importance, the reader is referred to the special sources of information.

Injury to the Spinal Cord.—*Complete Transverse Injury.*—In injury of the spinal cord, followed by complete anæsthesia, complete paraplegia, flaccid paralysis, and loss of tendon reflex, with vasomotor paralysis and absence of voluntary control of the bladder and the rectum, the inference is warranted that complete transverse destruction of the cord has taken place. The occasional presence, in these cases, of cutaneous reflexes or of twitching of paralyzed muscles from pyramidal-tract irritation, and the absence of the reaction of degeneration, need not cause a faltering in diagnosis.

Complete Unilateral Injury.—In complete unilateral injury of the cord, complete paralysis and loss of muscular sense occur on the same side as the injury, and all sensation except muscular sensation is abolished on the opposite side. The muscles energized by the injured segment of the cord undergo atrophy, and below this area spastic paralysis and increase in reflexes occur, and a zone of anæsthesia is located above the paralyzed area.

Partial Lesions of the Cord.—The manifestations of partial lesions of the spinal cord depend on the situation and the extent of the injury. An injury of the pyramidal tract may interfere with the voluntary control of the cerebrum over the ganglion cells below the seat of the lesion, and if the inhibition continue spastic paralysis will follow. Disturbances involving the anterior horn interfere with the transmission of impulses from the cerebrum to the periphery, destroying the reflex arc and causing flaccid paralysis. When individual ganglion cells or groups of cells are destroyed, the resulting secondary changes will correspond to the nerves and muscles directly associated with them. In a partial lesion of the pyramidal tract above the cervical enlargement the lower limbs are more paralyzed than the upper. Division of the

anterior roots will produce the same result as the destruction of the corresponding ganglion cells. A lesion of the posterior tract will cause ataxia and disturbances of muscular, tendinous, and joint senses of the same side as the injury, and the sense of touch may be abolished. In destruction of the posterior roots sensory and reflex phenomena are lost. A lesion of the posterior horn near to its base will cause disturbance of the pain and temperature senses. Destruction of the roots of certain cervical nerves will affect the arms only, the legs remaining normal. Individual muscles and sensory areas are fortified, as a rule, by a chief nervous supply, supplemented by a minor one at either side of this; therefore, paralysis may not be evident until after the three associated roots are destroyed. Injury involving the phrenic nerve may cause paralysis of the corresponding part of the diaphragm. Injury of the eighth cervical nerve or the first dorsal, or fracture of the corresponding vertebræ, or injury to the spinal cord relating to fracture of any of the dorsal vertebræ from the third to the sixth, and even higher than the sixth, may injure the ciliospinal centre of the cervical sympathetic, causing stimulation of the nerve with dilatation of the pupil, or paralysis of it with contraction of the pupil.

Vasomotor Paralysis.—In severe injury of the spine, vasomotor paralysis occurs, causing an increase of the amount of blood in the corresponding parts of the organism. The parts thus affected become warmer, the veins are distended, and priapism is likely to be present, especially in young males. The internal organs suffer, perhaps because of resulting anæmia; and the organs which suffer chiefly are the kidneys and bladder.

The Cerebrum, Etc.—The contents of the cranium are not considered in this article, except in the general isolated manner already stated. The extended scope and the special importance belonging to this topic render it inadvisable to consider the subject in this place.

Effects of Traumatic Lesions of the Spinal Cord from Disease or Injury (from Weichmann).—In this list the effects of traumatic section of the spinal cord through certain specially selected parts are illustrated only by a few of the more practical examples:

Fourth Sacral.—Paresis of the levator-ani, sphincter-ani, and the detrusor-urinæ muscles.

Third Sacral.—Paralysis of the preceding muscles, paresis of the rectum and bladder, loss of ejaculatory power, and weakened erection.

Second Sacral.—Loss of erection, plus the preceding results.

First Sacral.—Paralysis of anus, bladder, and genitals, etc.

Fifth Lumbar.—Paralysis of rectum, bladder, and genitals, etc.

Fourth Lumbar.—Paralysis of rectum, bladder, and genitals, etc.

Third Lumbar.—Paralysis of rectum, bladder, and genitals, etc. Loss of patellar reflex; ankle clonus may be present.

Second Lumbar.—Patellar, tendo-Achillis, and cremasteric reflexes lost; sensation of testicle lost, etc.

First Lumbar.—Patellar and cremasteric reflexes lost, tendo-Achillis reflex increased or lost, etc.

Twelfth to Third Dorsal.—Complete anæsthesia downward from a little below the seat of injury; complete paralysis; reflexes of lower extremity lost (exaggerated if lesion be incomplete); paralysis of respiratory muscles causes diaphragmatic breathing.

Second Dorsal.—Anæsthesia in a line with the second interspace; also at inner surface of the upper third of arm, plus the preceding.

First Dorsal.—Pupil disturbed; modifications in power and sensation of upper extremity and of the pectoralis muscles. Pronator quadratus weakened.

Eighth Cervical.—Upward increase of anæsthesia and increased involvement of the corresponding muscles; loss of digital abduction and flexion of little finger (difficult); pupil distended.

Seventh Cervical.—Pronation of forearm impaired or lost; supination possible; hyperæsthesia on radial side of arm, forearm, and hand; arm reflexes lost.

Sixth Cervical.—Increased upward paralysis and loss of sensation; difficulty in turning the head; reflexes of arm lost. The impairment of the respiratory function may soon cause death.

Fifth Cervical.—Complete paralysis of upper extremities; scapula can be raised; rotation and bending of head difficult; dyspnœa from involvement of phrenic nerve causing paresis of the diaphragm. Anæsthesia up to the lower part of the neck; death not long deferred.

Fourth to First Cervical.—Complete transverse lesion causes immediate death from loss of power of the diaphragm, due to destruction of the phrenic. In focal and unilateral lesions of this part of the cord life will be prolonged and recovery may take place.

The Genito-urinary System.—The attention of the reader is directed to the following subdivision of this topic:

1. The modifications of the normal excretory power of the kidney.
2. The modifications in the manner of passing the urine.
3. The modifications in the composition and character of the urine.
4. The examination of the urethra, prostate, bladder, ureters, and kidney.

Rarely is there anything of an interrogative nature in medicine or surgery of more importance than that of a careful scrutiny of the urine in all surgical cases, especially those in which an operation is required. Not one only, but repeated examinations should be made of the urine, of a thorough, searching character, and by one whose well-known competency in this respect admits of no doubt. The finding, in a single instance, of casts or albumin or of other objectionable factors ought not to be regarded of much greater significance

than that it necessitates a closer scrutiny, when possible, before the inauguration of operative action. However, when operative delay will not bide longer scrutiny, then action based on the full significance of the findings should be employed at once.

The amount of the urine passed in a given time, and its chemical composition as modified by the amount of fluid taken, are matters of supreme importance. It is in these circumstances that it is especially important to investigate the presence or not of œdema, high arterial tension, distinct atheroma, cardiac enlargement, etc. And if it happen that the patient have nausea, headache, respiratory distress, with contracted pupils and other well-known and dreaded evidences of renal disease, the question of what to do and when to do it is of no small moment. In these circumstances even a cautious administration of a general anæsthetic, an insignificant operative effort, or any act that produces some depression of the patient's strength may be promptly followed by suppression of urine and death. The easy, painless passing of a sound has been known to precipitate the final act. Heedless attention and incomplete preparations in operations in these cases have cost many lives and cast many shadows on otherwise justifiable operative endeavor.

The modifications in the manner and frequency of passing urine are the outcome of changes in the channel, in the size and shape of the stream, and in the urgency of the act. The free flow and the full oval stream of health are hindered, thinned, or made dribbling by constriction or obstruction of the channel, by narrowing of the orifice, or by loss of the expulsive force. Therefore stricture of the canal or the presence of gravel in it, or narrowing of the meatus from inflammation, ulceration, or some congenital defect, and loss of the effect of expulsive power of the bladder from paralysis or obstruction, are among the causes of a deformed stream.

Frequent micturition, from intolerance of the bladder to the presence of urine, or from the presence in it of a stone, a foreign body, or a morbid growth, is quite common. Diseases of the spinal cord and tumors of the medulla so modify the reflex centre of the organ as to hasten the frequency of micturition. All varieties of cystic inflammation, prostatic enlargement, changes in the specific gravity and constituency of the urine dependent on diabetes, oxaluria, etc., and cantharidal medication, hasten the act. Renal and ureteral calculi and diseases of the rectum, anus, and spinal cord add urgency to the intent.

Diminished frequency of micturition is less common than the former act. An abeyance or loss of the normal sensation of the mucous membrane that signals the presence of urine in the bladder, by reason of the local numbing effect due to a fever, to cerebral disease, to alcohol, opium, etc., to say nothing of indifference to the desire, diminishes the frequency of urination. Free perspiration, the ingestion of a small amount of fluid, spinal concussion, delayed

metabolism, and diminished excretion from kidney disease accomplish the same result.

Retention of Urine.—Retention of urine may be either incomplete or complete. Retention is dependent either on obstruction of the channel or on diminished expelling force, or on both combined. Retention of urine in severe injury of the spine is the result of reflex contraction of the vesical sphincters, dependent on the influence exercised by the nerve plexus in the wall of the bladder, when the spinal centres are at fault. Prostatic disease, pedunculated tumors of the neck of the bladder, or foreign bodies or blood-clots in that cavity cause retention by obstructing the inner orifice of the urinary channel. Inhibition of the lumbar centre from shock, operations on the urinary organs, rectum, and contiguous parts, often cause retention, requiring the use of a catheter to relieve the distress. Overdistention, voluntary or otherwise, causes retention of urine; and it should be said at this time that in this condition the bladder should not be emptied at once, but partially emptied instead, thus avoiding the collapse and paralysis of the bladder, with the congestion and possible inflammation of the organ that often follow a prompt emptying.

A too great emphasis cannot be laid upon these facts. Complete retention is infrequent, as a few drops escape from time to time in nearly all instances. Complete retention of urine may be mistaken for rupture of the bladder and for suppression of urine. Partial retention is often mistaken for involuntary and frequent micturition, as it is denoted by frequent urinary acts, feeble stream, and perhaps by dribbling of urine. The introduction of a finger into the rectum or of a catheter into the bladder will determine a difference between complete retention and rupture in the former, and partial retention and involuntary, frequent micturition in the latter instance.

Overflow of Urine.—This expression signifies that there is retention as well as overflow of urine. The bladder becomes much distended and the urine escapes periodically, attended often by painful vesical contractions. Later, however, paralysis of the bladder ensues from the effect of long-continued distention or overdistention, and dribbling of urine is the result. These cases have been mistaken for frequent micturition. A means of differentiating the two conditions has already been mentioned.

Irrepressible Micturition.—In this form the bladder, the nervous supply, and possibly the urine itself are concerned. The diseases that—in co-operation, perhaps, with an ultra-irritating urine—increase the sensibility of the mucous membrane of the bladder and its nervous supply, exalt the urination sensation to such a degree as to render proper control of the act impossible. In acute cystitis this form of urination is of common occurrence.

Urgent Micturition.—In urgent micturition the desire to urinate is strong, yet controllable. This form is more frequent than irrepressible micturition.

Preoccupation, suggestive sounds, as of falling water, mental emotions, as apprehension and fright, may each prompt the desire.

Retarded micturition is characterized by the unusual length of time occupied in completing the act. The retardation may be due to delay in "starting the stream," or to slowness in emptying the bladder, or to a combination of both of these causes. This kind of urination does not call for active interference on the part of the surgeon. As a matter of fact, the presence of this condition is appreciated only when the time available for a special completion of the act of urination fails to accomplish the purpose. Delay in "starting the stream" comes from slight obstruction to the passage of the water or from slowness of action on the part of the expulsive forces. Atony of the bladder, special nerve lesions, mental emotions, and blunted sensibility, etc., contribute each something toward the production of this condition.

Interrupted Micturition.—The normal act of urination is free and continuous until near the completion, when spasmodic acts cause the escape of the urine in jets, with or without interruption of the flow. The causes of the interrupted variety of urination are to be sought for in the bladder and urethra, and its mechanism is largely of a valvular type, the force of the stream effecting the closure. Stone, blood-clots, pus, stringy mucus, pedunculated growths, foreign bodies, etc., are common examples of the obstructing agents. Congestion of the prostate or of a urethral constriction often causes matutinal interference with the stream. The causes of interrupted micturition often lead promptly to difficult micturition.

Difficult Micturition.—Difficult micturition is often associated with and follows the interrupted kind. Anything which weakens the powers of expulsion, especially the bladder, or obstructs the flow of urine, is likely to cause difficult micturition. When the difficulty always occurs at the beginning of the act, urethro-vesical obstruction is indicated; when it occurs at the close of the act, stone, blood-clots, and foreign bodies in the bladder are causes of the difficulty. The loss in the contractile power in the bladder from any cause occasions this kind of trouble.

Obstructed Micturition.—Obstructed micturition follows an advanced state of the disorders which cause difficult micturition. In practice it is not wise to make a special distinction between the obstructed and the difficult kinds of urination, since the earlier the treatment of the cause the safer it is for the patients and the better are the results.

Incontinence of Urine.—Incontinence of urine signifies the inability of the bladder to restrain the escape of its normal contents, the urine running away as soon as it reaches the bladder, unless detained by the force of gravity or the friction incident to the curves of the urethra. The above statement defines "true" incontinence. The expression "false" incontinence is misleading and nosologically inaccurate, since it corresponds only to the involuntary and

unconscious acts, better and correctly expressed as "overflow" of urine. Incontinence depends on any modification of the bladder which permits early, continuous, and uninterrupted flow of urine from it, rendering the bladder a passive part of the urinary canal. Prostatic hypertrophy, interfering with the vesical sphincters; malformation of the bladder; paralysis of the neck of the bladder and of the sphincter muscles of the urethra, are causes of this affliction.

Involuntary Micturition.—Involuntary micturition in adults is an outcome of both hyperæsthesia and anæsthesia of the bladder. In children various reasons are assigned for this infirmity, among which may be mentioned irritation of the anal and urethral openings and insufficiency of the vesical sphincter, allowing urine to enter the urethra, from which it is promptly expelled. In adults hyperæsthesia is encountered in those who suffer great hardships and deprivation, and are afflicted with those pathological conditions which commonly produce an irritable bladder. In those afflicted with typhoid and typhus fevers, and in profound asthenic states from other causes, the anæsthetic variety is the one commonly observed. Also fright and shock often cause involuntary micturition.

Painful Micturition.—Discomfort and pain of the urinary tract occur *before* micturition, on account of the effects of causes that increase the sensibility of the mucous membrane of the bladder and the prostate and the irritating power of the urine. Therefore the causes of these two factors of the trouble are indeed numerous. Pain *during* micturition depends on diseased action within the bladder or within the urinary canal. Cystitis and urethritis from various causes, increased irritating changes in the urine, are the fertile sources of this form of infliction. Pain *after* micturition may be of two kinds; it may either increase or diminish in severity after the completion of the act. The diminution of pain after micturition indicates that contact of urine with the bladder and urethra, or distention of the bladder, was the probable cause of the pain. An increase of the pain *toward the end of micturition* and *after the act* warrants the diagnosis of stone in the bladder or of enlarged and hypersensitive prostate from various causes. In this variety of the disorder the emptying of the bladder of urine permits the organ to contract on the stone or foreign body, or on the enlarged and sensitive prostate, causing severe pain in the bladder and at the end of the penis, or possibly in the latter only. In micturition, pains referred to the thigh, testicle, or loin originate not infrequently from the pelvis of the kidney and from the ureter; those in the sole of the foot, calf of the leg, and thigh frequently depend on urethral stricture. Referred prostatic pains appear in the perinæum and lower part of the rectum. Hence, the local and referred pains of micturition should be given a careful study.

Force of the Stream.—The force of the stream may be increased or diminished. The causes of urgent and of irrepressible urination increase the force

of the stream. The force is diminished by the various causes of weakened vesical contraction and by the presence of obstructive influences in the urinary canal.

The Consideration of the Genito-Urinary Organs.—This is not the proper place in which to consider the various diseases of the urethra, but we may be permitted to say a few words with regard to the relation of the urinary canal to the perinæum and the rectum, as bearing on the important subject of urinary extravasation. The perinæum should be examined, both deeply and superficially, in order properly to interpret the changes that arise from urinary extravasation at this situation. The deep early induration incident to rupture of the membranous portion of the urethra—an induration which is felt in front of and from within the anus—contrasts strongly with the more extended and superficial induration which follows an escape of urine either from a deep-seated rupture or from a rupture of the anterior portion of the urethra. It is a matter of great practical importance that the surgeon should be thoroughly familiar with the anatomical relations of the different structures in this region: the bulb of the urethra, the median line of the perinæum, the rami of the pubes and ischium, and the tuberosities of the latter.

The Prostate.—The condition of the prostate in health and in disease can be estimated by the finger introduced into the rectum. The size, shape, and sensibility of the organ may thus easily be ascertained. The sensibility of the structure and the deviations of the prostatic sinus can also be learned by means of suitable instruments introduced into the urethra. Consequently it is an easy matter to determine the presence of prostatic enlargement and to ascertain with reasonable certainty the nature of obstructive changes in the prostatic urethra. The relations between pain and urination in various kinds of prostatic interference with the act have been given already as full consideration as our space will permit. It is necessary to remember, however, that a sensitive prostate and the presence of a stone in a sensitive bladder cause similar pains after urination, and for similar reasons; also that a movable stone in the bladder arrests the urinary flow promptly, and that a change in the position of the patient may as promptly relieve the pain; and, finally, that an enlarged prostate slowly diminishes the flow, which is not influenced by change in position. With the finger in the rectum the seminal vesicles can be felt high up, on either side of the prostate. At the same time the soft area of a prostatic abscess, the hard area of a prostatic calculus, the hard and irregular nodular outlines of malignant and tuberculous disease, may also be determined.

The Bladder.—The relative situation of the bladder in the pelvis of the different sexes, different ages, and at different periods following micturition is exceedingly important in cystotomy and puncture of the bladder above the pubis; also in the instances of external violence directed from above into the

pelvis (wagon wheel, kick, etc.) or in fracture of the pelvis. In these circumstances the greater the amount of urine in the bladder the greater is the danger of rupture, and conversely. The outline of an overdistended bladder and the outline of an enlarged uterus are each of prime importance, and often of embarrassing and disastrous significance when mistaken for each other. The history of a long interval in urination, attended by the development of an oval, suprapubic, abdominal tumor, disappearing coincidently with violence of any kind or without apparent cause, should arouse serious apprehension and corresponding activity regarding rupture of the bladder. If, however, the bladder has become contracted, because of disease or obstruction of the urinary channel, less distention and correspondingly less danger will attend the case in these circumstances.

The bladder can be examined by means of the finger introduced into the rectum; and it can be examined from the inside by the aid of various devices constructed for that purpose (consult the article on Surgery of the Bladder, in a later volume). A certain amount of information can also be obtained by percussion, by palpation through the overlying abdominal wall, and by combined palpation and the use of the x-ray. By means of the finger introduced into the rectum one can determine the presence of a collapsed or distended bladder (useful in detecting rupture of the bladder), of a sensitive or insensitive organ, of the presence in it of a foreign body, the extremity of an instrument, or a solid growth; and especially are these features emphasized by combined manipulation. A knowledge of the points of reflection of the peritoneum from the bladder should be known, so that a proper estimate may be made of the question whether or not extraperitoneal or intraperitoneal rupture of the organ has taken place. Combined manipulation, with the hand on the abdomen and an instrument in the bladder, may be practised, but always with exceeding caution. As I am convinced, from personal knowledge, that errors with regard to this matter are of frequent occurrence, I will again caution against the mistaking of the involuntary act and the overflow that attends an overdistended bladder, for unobstructed, frequent micturition. The x-ray may reveal the presence of a stone or a foreign body in the bladder, or the existence of a fracture or a dislocation of the pelvis.

The Ureters.—The condition of the ureters can be ascertained by means of external manipulation, by cystoscopy, by direct catheterization of these channels, and by explorative incision.

In thin subjects and in those with much thickening or distention of the ureter from inflammation, from an accumulation of urine, from the presence of a stone, or from any other cause, external palpation may serve to locate the tube, especially the lower part, provided vaginal or rectal palpation be added to the abdominal effort. In stout subjects, on the other hand, external palpation can offer no encouragement; at the most, it may enable one to ascer-

tain the presence of a tumor or of tenderness at or near the site of the ureter. However, by the aid of cystoscopy, supplemented with catheterization of the ureters, it is possible to determine whether or not the canal be permeable or be obstructed with stone or inflammatory products, and whether it be discharging healthy or abnormal urine. The latter fact can be made out by the cystoscope alone, and thus the state of each of the kidneys may be decided. Segregation of urines may be practised for a similar purpose. Increased frequency of urination attends pain in the ureter, especially when it is due to a passing calculus. The kidney also may suffer from referred pain in these cases.

The Kidney.—A kidney is said to be *movable* when the entire length can be examined, and to be *floating* when it can be freely moved in any direction. Misplacement of the kidney happens about once in one thousand cases. The misplacements may be slight or excessive, or of little degree, and the misplaced organ may occupy the iliac fossa or may lie against the promontory of the sacrum or between the rectum and bladder, etc. Sometimes both kidneys are misplaced. A single kidney happens once in twenty-four hundred autopsies (Morris). In the absence of a kidney the opposite one is much increased in size. In a physiologically enlarged kidney the normal outlines of the organ are maintained. In enlargement from morbid causes, as from distention or from a tumor, the outline will be changed, but the modification will vary according to the location, the extent, and the nature of the disease. Overdistention and acute malignant changes obliterate the normal outlines of the organ, often fusing them with those of the contiguous soft structures. As a rule, the inner outline maintains the longest a distinguishing feature—the notch indicating the hilum. The right kidney may be mistaken for a distended gall bladder, the left for the spleen. The presence of the colon in front and on either side of the kidney, with tympanitic resonance when the intestine is distended; the greater freedom of movement of the gall bladder on manipulation or when the position of the patient's body is changed, unless the gall bladder be adherent —in which case, however, it still will be more movable than the kidney; the superficial location of the gall bladder as contrasted with the deep position of the kidney, with often a marked interval between them, discoverable when pressure is made upon the gall bladder—these are the important diagnostic features which should be borne in mind when an examination of the kidney of the right side is made. On the left side the shape and the location of the spleen, its movements on respiration, the sharpness of its border, and the fact that it is in front of and the kidney behind the colon, are the diagnostic features of importance. So far as the kidney is concerned, the following favorable facts should be remembered by the surgeon: the relation of the organ to the peritoneum; its accessibility from the loin for operative purposes; and its possession of a fatty capsule.

Finally, there remain to be mentioned the following common manifesta-

tions of morbid action on the part of the genito-urinary organs: the connection of priapism with injury of the spinal cord, the retraction of the testis in renal colic, the elongation of the prepuce in stone of the bladder, urinary incontinence, and local itching.

The Employment of a General Anæsthetic for Diagnostic Purposes.

The surgeon may often be very greatly aided in his efforts to make a diagnosis by administering to the patient a general anæsthetic. This procedure not only renders the manipulations painless, but also causes complete relaxation of all the muscles, and thus renders it possible for the surgeon, in an obscure case—*e.g.*, an abdominal tumor or an injured hip or elbow—to determine the outlines and nature of the tumor, and to ascertain the character and full extent of the injury. The fright of children and the serious apprehension of adults in regard to pain, as well as their sensitiveness about having their private parts exposed, may be thus entirely relieved. It should be remembered, however, that relief from pain by this means does not give the surgeon license to exercise unnecessary or unduly prolonged or needlessly severe efforts in attaining the purpose. On the contrary, great discretion should be exercised in these circumstances, and often actual restraint is needed on the part of the surgeon to prevent the adding of increased hurt to previous injury, more especially in the case of contentious persons. Only sufficient force, and that under intelligent guidance, should be used to achieve the aim in view. The giving of an anæsthetic only for the purpose of eliciting crepitus in a fracture that should be determined by other means, is a use of opportunity that can be seldom justified. The rupture of an abscess by the employment of a degree of force which it is difficult to estimate under these circumstances is a result deeply to be regretted.

Local anæsthesia of rectal and other passages is sometimes employed to lessen the infliction of pain during an examination. The employment of atropia in the eye, of tuberculin for tuberculous invasion, of specific treatment as a test of syphilis, of pilocarpine to test the response of the cervical sympathetic, of cathartics to evacuate the bowels, are illustrations of the use of drugs in diagnosis. The fact that opium masks symptoms, and may for this reason mislead the surgeon, should be kept in mind.

Operative procedures of a simple or severe nature are employed for diagnostic intent, and always the strictest aseptic technique should mark the performance. The use of the trocar and cannula, the aspirator, the aspiratory needle, and the hypodermic syringe to determine the presence, the situation, and the nature of a morbid process related to various parts of the body, is already well understood. The danger of infection from the escape of fluid, especially into the peritoneal cavity in the absence of adhesions; the danger of

infecting the healthy skin from puncturing an underlying malignant growth, and of the involvement of important overlying tissues in instances of operative approach, are illustrations of danger demanding thoughtful care. The puncturing of the thorax, the abdomen, and the bladder are usually innocent procedures, but of sufficient import to exact exceeding care in their use. The incision of a tumor for diagnostic purposes, followed by its removal at once if the conditions found demand it, or by closure of the wound if further operative interference be found unnecessary, is sometimes practised in tumor of the breast and elsewhere. Malignant infection of the healthy parts is regarded as possible in this measure. Abdominal explorations should not be done solely for the purpose of making a diagnosis, but with the further idea of gaining additional knowledge regarding the prognosis and treatment of a case. Therefore, before making an explorative incision, one should stop to consider the advantages to the patient that may follow the act. The practice of making an explorative incision to determine only a question in prognosis is unjustifiable, except when requested by a patient having a full appreciation of the important facts in the case. The cutting into an inoperable malignant tumor can rarely be justified, as no physical gain can follow and decided loss is almost sure to result. The idea that an explorative incision is devoid of danger has cost many lives, saddened many hearts and homes, and impaired many professional reputations.

Editors' Note.—The article on Blood Pressure, etc., referred to on p. 533, was to have been published in the present volume, immediately after that on Surgical Shock, but the sudden illness of the author has compelled us to transfer it to the Appendix, at the end of vol. viii.

THE BODY FLUIDS IN GENERAL SURGICAL DISEASE, WITH SPECIAL REFERENCE TO THEIR DIAGNOSTIC VALUE.

By HARLOW BROOKS, M.D., New York City.

Although the study of the body fluids has been largely developed in connection with surgical science, the discussion of this subject is now more extensively treated in works relating to internal medicine. This is not as it should be, for the well-equipped surgeon must have at his command all the facts and methods of medical study, since in so many instances surgery, both in diagnosis and in treatment, is now called upon to assist or supersede the methods of internal medicine.

Since the space at our command is so limited, it has seemed best to me to consider the subject entirely from the practical rather than the theoretical standpoint, and for this reason I shall devote myself chiefly to matters of diagnostic importance, for it is in this direction that study of the body fluids has proven of greatest utility to us.

References to technique and methods of examination have been omitted, for the reason that space does not permit of their full statement, and incomplete discussions of technical matters are more misleading than they can possibly be useful. Full elaboration of this subject is furnished in such special and general text-books as those of Ewing, Wood, Simon, and Mallory and Wright. But very little reference will be made to the characteristics of the normal fluids of the body, as it is assumed that the reader is already familiar with them.

Although the data secured from the study of the body fluids often appear to be of the most conclusive nature possible, in diagnosis particularly they should never be considered absolutely final, but must be accorded the value of symptoms only.

There is now perhaps a tendency in modern medicine to overvalue, as in the past the inclination has been to undervalue, the evidence of the test tube and the microscope; but the broad-minded clinician must first secure all the evidence at hand, and then with careful judgment eliminate the unimportant, until final diagnosis rests not on any one sign or symptom, but on all.

THE BLOOD.

The examination of no one tissue yields more valuable data in general surgical diagnosis and prognosis than does that of the blood. This examination may be special, brief, and very limited in its extent; or in other cases, where a broad and comprehensive view of not only the special condition but also of the

general nutritive functions of the body is desirable, a detailed study may be necessary, and will richly repay the time spent on it.

A great deal of important evidence can be elicited even by the most simple and primitive examination. Thus, the puncture of the finger-tip or lobe of the ear, with close observation of the exuding drop, judged from the rapidity of the flow, may give the required data as to the relative abundance of blood fluids. In other instances, where it is desirable to ascertain perhaps the coagulability of the blood as a preliminary to operation, tolerably definite information may be thus obtained by timing the clotting of the drop as it flows from the minute wound, though this can be determined with absolute accuracy and almost as easily by the use of the extremely simple and ingenious coagulometer of Biffi. In cases suspected of hæmophilic tendencies, this same simple observation discloses a condition the knowledge of which may save the operator serious difficulties. In this disease it is well for us to recall that, in at least some cases, the blood, when removed from the body, clots in very nearly the normal time, as in a case recently reported by me; the primary essential lesion in hæmophilia being not in the blood, but in the blood-vessels. Clot formation is notably delayed in many, but not all, cases of jaundice, particularly when the hæmoglobin percentage is low; and this should be taken into consideration when operation on jaundiced and anæmic patients is contemplated. Recent hemorrhage is suggested by increased fibrin formation, and, when this factor has been watched throughout the course of the disease and a rapid increase is discovered, it is strongly indicative of hemorrhage, perhaps from obscure and unsuspected foci. Even the presence or absence of anæmia of marked degree may be roughly suspected by this method, although in all cases of this nature a thorough examination of the blood should be made.

The percentage of hæmoglobin is often a measure of the gravity or of the duration of the disease process, and it is therefore specially important to ascertain this when the question of operation arises; for it is much better in many cases, in which immediate operative relief is not demanded, to wait until a higher percentage can be secured, not only because both chloroform and ether greatly depress the hæmoglobin content, but also because in cases of this nature convalescence is prolonged and even the chances of ultimate recovery may be jeopardized. This seems particularly true in cases of chlorotic anæmia, in which, as a rule, preparatory treatment of the anæmia, for even the brief period of a few days, greatly improves the general condition of the patient.

A knowledge of the hæmoglobin percentage is often of assistance in the differential diagnosis of malignant and innocent neoplasms, for, as a rule, it will be found that the percentage either diminishes rapidly or remains stationary at a low figure in the malignant tumors, while severe or progressive anæmia is rare in innocent growths, except when it is due to loss of blood or to some other more or less independent pathological condition.

EXPLANATION OF PLATE II.

(Preparations Stained by Goldhorn's "One Solution.")

Fig. 1.—Myelogenous Leukæmia. The patient was a man 42 years of age. He presented himself on account of an abdominal tumor, which was found to be a greatly enlarged spleen. Lymph nodes but slightly enlarged. The case presented but slight anæmia, and its real nature was not suspected until the blood was examined.

The cut shows the presence of frequent very large mononuclear white corpuscles (myelocytes), the cytoplasm of which is studded with neutrophilic granules. The presence of these myelocytes typifies leukæmia of this special variety.

Fig. 2.—Severe Secondary Anæmia, Clinically Simulating Pernicious Anæmia, but Found to be Due to Chronic Atrophic Gastritis. Hæmoglobin, 53%; red corpuscles, 2,004,000; leucocytes, 6,150.

The illustration shows marked variation in size and deformation (poikilocytosis) of the red corpuscles. The amount of hæmoglobin contained in the cells is very irregular; some of the cells are deficient in hæmoglobin, while others contain more than the normal quantity. Polychromatophilic or granular degeneration is shown in two large red cells (macrocytes). Complete recovery, in so far as the blood condition was concerned, followed appropriate treatment.

Fig. 3.—Lymphatic Leukæmia. Male, aged 47 years. Enormous enlargement of superficial and deep lymph nodes, liver, and spleen. Slowly progressive enlargement of lymph nodes extending over the past seven years, first noted in superficial cervical nodes.

The figure shows a typical microscopic field illustrating the marked relative and absolute increase in the lymphocytes. The preponderance of these cells characterizes this leukæmia and distinguishes it from the myeloid form shown in Fig. 1. Two myelocytes are present in the field. The case presented profound anæmia (Hæmoglobin 42%), as indicated by the very pale red corpuscles, a few of which, however, show an abnormally high hæmoglobin index.

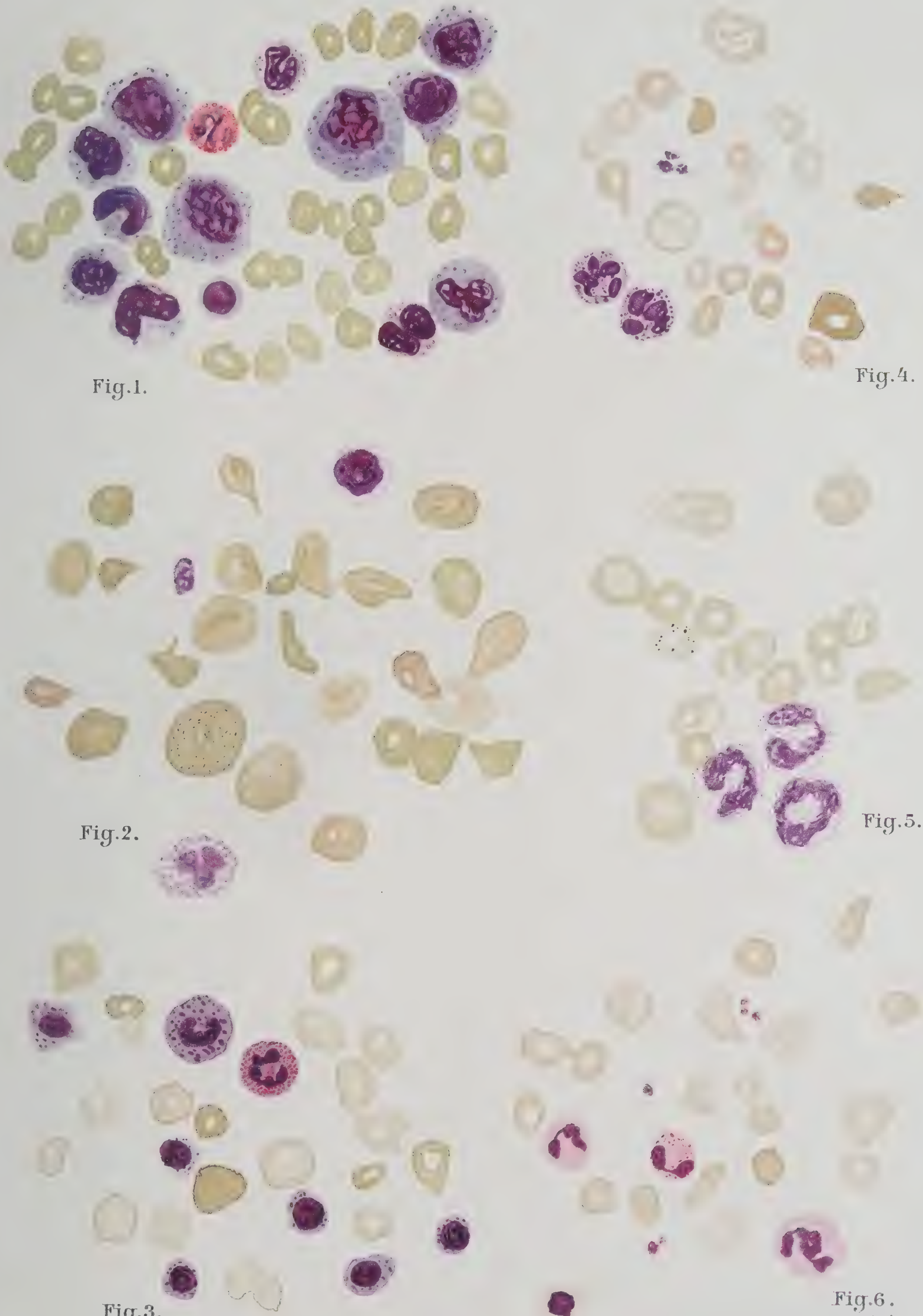
Fig. 4.—Severe Secondary Anæmia, Following Long-continued Hæmorrhage (Epistaxis and Menorrhagia). Hæmoglobin, 30%; red cells, 3,112,000; leucocytes, 9,500. Note the deformity (poikilocytosis) and the low hæmoglobin index of the red corpuscles. Some of the corpuscles show beginning endoglobular degeneration.

Fig. 5.—Anæmia Five Days After Severe Hæmorrhage. Traumatic rupture of the spleen with splenectomy. Complete recovery.

The blood shows variation in hæmoglobin index, a few macrocytes, slight endoglobular degeneration and occasional polychromatophilic degeneration of the red corpuscles. A slight polynuclear leucocytosis was present in this case.

Fig. 6.—Fatal Anæmia Terminating Mixed Intestinal Infection with the Hook Worm (Uncinaria americana), and a Tape Worm (Tænia saginata). The case was originally diagnosed as one of duodenal ulcer.

The figure shows the marked degeneration and deformation of the red corpuscles; endoglobular changes are very marked in the average red cell. One nucleated red corpuscle (normoblast) is present. The eosinophilia, usually marked in cases of this kind, was but slightly developed in this instance (3% to 5%), probably on account of the lack of reaction on the part of the greatly depressed body tissues.



Harlow Brooks Del.

Lith. Anst. v. E. A. Funke, Leipzig.

Plate Illustrating Various Blood Lesions.

Whenever anæmia occurs in any surgical disease, its nature, whether primary or secondary, and its cause should always be sought for. In most instances this necessitates a count of the red blood corpuscles, which adds more than enough valuable data to repay the small expenditure of time necessary for carrying out this reasonably simple procedure. It is in this way, and also by the determination of the hæmoglobin percentage, that the differentiation between primary (Plate IV., Fig. 2) and secondary anæmia is made. Furthermore, in those cases in which disease of the internal viscera is suspected, it often directs the queries of the surgeon in the right direction. Even more important than the count of the red cells is, in many cases, the examination of these bodies when properly prepared and stained by one of the modern polychrome methylene-blue methods. For example, cachectic anæmia (Plate II., Fig. 2, and Plate III., Fig.

Fig. 142.—Ova and Embryos of Filaria immitis; from the Blood of an Infected Sea Lion.

1), in which one sees marked poikilocytosis, polychromatophilic and other degenerative alterations in the red corpuscles, may be distinguished from the anæmia of a person who is convalescing from an acute hemorrhage by the characteristic picture of pale red cells and numerous normoblasts which the latter condition presents (Plate II., Fig. 5). Malignant growths may also be thus in part differentiated from those of an innocent nature. In my opinion, careful study of the red blood cells, of their size and shape, of the presence or absence of nuclei, and of the changes in their cytoplasm, is, in the majority of cases, more instructive than the simple red-cell count with which so many clinicians are content. Malarial, trypanosomatous, or relapsing-fever infections discovered in this direct and absolute way may fully account for an otherwise confusing pyrexia or a splenic tumor (Plate III., Figs. 2 and 3); in the same way the demonstration of filaria (Fig. 142) may account for chyluria or elephantiasis. If

we find the picture of leukæmic blood, so patent to the merest glance, we may often be warranted in excluding the possible diagnosis of lympho-sarcoma or of tuberculous or syphilitic lymphadenitis (Plate II., Figs. 1 and 3). The presence of megaloblasts and normoblasts, in combination with an appreciable diminution in the number of red cells and a relatively high hæmoglobin index, may, in a case in which the other aspects seem to point rather to a gastric or an intestinal neoplasm, indicate a primary pernicious anæmia (Plate IV., Fig. 2). Probably, however, the information afforded by a determination of the hæmoglobin percentage, by a count of the red blood cells, and by an examination of the condition of these cells, is, from the standpoint of general surgery, the most important of all. It throws light upon the general nutritive conditions of the body as a whole, and indicates accurately the extent and the duration of the disease.

The surgeon should not content himself with a single examination of the blood or even with a few such examinations; he should—if he desires to throw light upon the diagnosis or to watch intelligently the progress of the disease—have the examinations made regularly and the results charted in the same manner as are the pulse and body temperature.

Of especial value to the surgeon is that part of the examination of the blood which relates to the counting of the leucocytes. By this means it is generally possible definitely to recognize the existence of a deep-seated inflammatory process which might otherwise escape detection. Sepsis and general as well as local infections are also manifested by the same procedure, at times with surprising accuracy (Plate IV., Fig. 3). The absence of leucocytosis is also of very great importance, since it either indicates that inflammatory lesions are lacking altogether or demonstrates the overwhelming virulence of some infectious process on the organism; furthermore, it may signify some special type of inflammatory disease, as tuberculosis, influenza, or typhoid, in which conditions the leucocytes are sometimes subnormal in number.

Much is also to be learned from a study of the types of leucocytes present in each case, and in either hyper- or hypo-leucocytosis differential leucocyte counts should be made. Thus, the diagnosis of lymphatic leukæmia may be made in large part from a relative and absolute increase in the lymphocytes. Trichinosis, hook-worm (Plate IV., Fig. 1, and Plate II., Fig. 6), and some other varieties of parasitic disease may be strongly suggested by proportionate increase in the eosinophilic white cells, which, it should be remembered, are also increased in certain forms of bone disease, notably in involvement of the marrow by some form of new growth. Relative increase of the polynuclear neutrophiles with leucocytosis is indicative of inflammatory disease, ordinarily of bacterial origin, and no leucocytosis should be taken as confirmatory of inflammation unless it be of this variety (Plate IV., Fig. 3).

The clinician must always bear in mind the frequent exceptions which are encountered in connection with leucocytosis. Thus, even large pus accumula-

EXPLANATION OF PLATE III.

(Preparations Stained by Goldhorn's Polychrome Methylene Blue.)

Fig. 1.—Cachectic Anæmia, from a Case of Uterine Carcinoma. The severity of the anæmia is shown by the decrease in number of the red corpuscles, their diminution in size (microcytosis), and poikilocytosis. Most of the cells show a high color index, causing the condition somewhat to resemble that seen in pernicious anæmia. A single myelocyte is present in this field, as is frequently the case in anæmia from malignant neoplasms. Hæmoglobin, 50%; red corpuscles, 1,430,000; leucocytes, 7,300.

Fig. 2.—Severe Secondary Anæmia due to Æstivo-autumnal Malarial Infection. The infection was contracted in Porto Rico during 1898, and resulted in death shortly after the patient's return to this country.

The severity of the infection is indicated by the number of invaded corpuscles seen in the single field. Five cells show small "ring-form" parasites; in one, two plasmodia are present. A single extra-cellular organism, a "crescent," is shown. The marked endoglobular degeneration of even the uninfected red cells is well indicated in the figure.

Fig. 3.—Double Tertian Malarial Infection. The double character of the infection is shown by the presence of two parasites nearing segmentation, and two relatively young forms. Paroxysms were quotidian in character. The blood also shows a moderate degree of secondary anæmia, indicated by poikilocytosis and low color index.

Fig.1.

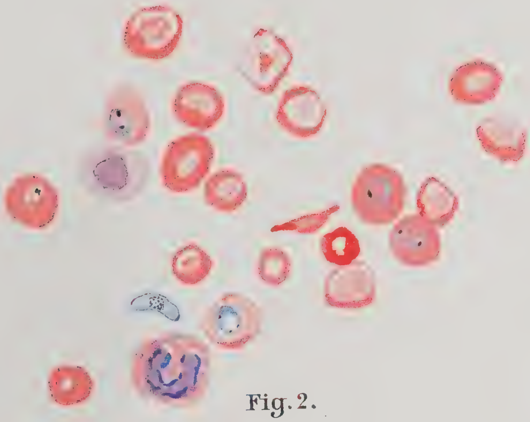


Fig.2.

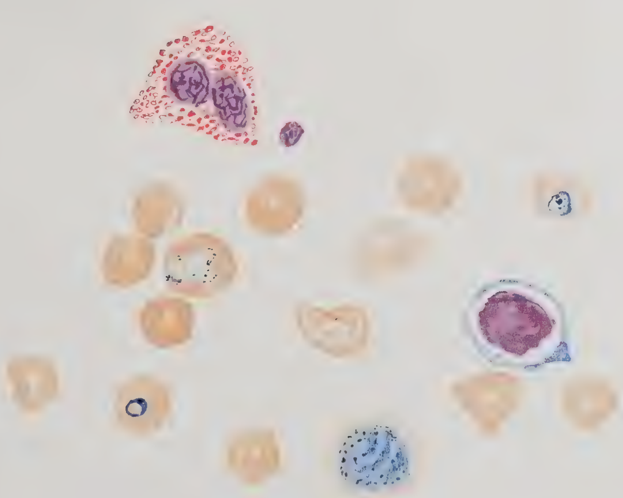


Fig.3.

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Plate Illustrating Various Blood Lesions.

tions, if of long standing or well encapsulated, may cause slight or no increase in the leucocytes, while occasionally very slight infections, as small boils, may, in particularly susceptible patients, cause a pronounced leucocytosis. In no important case should the surgeon be content with a single count, but several should be made and at different periods, so that technical errors and physiological conditions may not confuse the symptom.

Perhaps it is also well to point out the possibility of over-valuing leucocytosis as a sign in diagnosis. The surgeon may easily be led to draw wrong inferences if he fails to remember that leucocytosis, even of considerable degree, may exist as a physiological phenomenon, and that it may also follow the use of certain drugs, as phloridzin.

When there are found, in the blood, leucocytes the cytoplasm of which contains glycogenic granules that assume a dark-brown color when brought in contact with a solution of iodine (a condition to which the term "iodophilia" is often applied), we may interpret this circumstance as indicating quite accurately that pus has formed somewhere in the body. On the other hand, it is not safe to infer that the absence of leucocytes of the variety we have just described may be taken as an evidence that pus has not formed in any part of the body. It must also be remembered that diffuse amyloid degeneration, such as is observed particularly in syphilis and in tuberculous or chronic inflammatory disease of bone, is likely to be characterized by the presence, in the blood, of these same leucocytes. When the iodophilia is found to be progressive we are warranted, I believe, in drawing the inference that the suppuration is also on the increase.

The relation of the blood plates to surgical diseases I believe to be very indefinite and uncertain. An increase in the number of the platelets is taken by some as indicative of a tendency to rapid clot formation. A few authors think that these bodies are actively concerned in the thrombosis so frequent in certain diseases, particularly in pneumonia and typhoid. A close study of a large series of cases in regard to this point during the past two years has fully convinced me that there is no relationship between the number of blood plates and the tendency to thrombosis or rapid clot formation.

Bacterial examination of the blood is resorted to more and more frequently in cases in which there is question as to general hæmic infection, and its great utility cannot be overestimated. It sometimes affords the only means at our disposal for demonstrating what particular bacterial agents are concerned in any general and in certain local infectious processes. It should be generally understood that the amount of blood required for a satisfactory examination should be relatively large, that the amount of the media into which the blood is to be inoculated should be abundant, and, finally, that several kinds of pabulum should be used, particularly such as most closely approximate the human serum. Negative results in these examinations are not to be given too much consideration in diagnosis, on account of the great possibility of technical errors. Posi-

tive findings are of the most definite character possible, and in a good many instances this demonstration of the etiological factor of the disease shapes not only the diagnosis, but also the treatment and the prognosis.

Kryoscopy of the blood is as yet ordinarily but little employed in general surgical diagnosis or study, though its findings are occasionally of great worth in special surgery, particularly in diseases of the kidney or in cases in which transudates are formed. Kryoscopy of the urine or of the transudates or exudates is of little utility unless compared with the basis furnished by the same method applied to the blood. When this method is employed for the blood alone it does not appear to furnish as trustworthy results as are supplied by other and better-established methods.

The determination of the alkalinity of the blood, though often of interest and value to the internist, particularly in such conditions as diabetes or in the severe anæmias, has thus far, in my hands, proven of little value as an assistance in surgical diagnosis.

The more unusual methods of examining the blood—such, for example, as those for determining the total percentage of iron and the specific gravity—have thus far proved of little use. This is doubtless largely due to the facts that the technical details are somewhat complicated and consume considerable time, and that we do not yet possess sufficient physiological data on which to base our clinical studies. There can be no question that, as the normal chemistry of the blood becomes more thoroughly elucidated, the pathological chemistry will keep pace and will finally yield results probably more valuable even than are supplied by our now largely morphological studies. The very suggestive work on the psonins, toxins, and antitoxins promises much for future research along these lines.

The serum reactions which are based on the formation of specific anti-bodies in the blood under the influence of specific infections or toxæmias, while they are more commonly employed in medical conditions, also lend themselves to surgical diagnosis with equally beneficial results. The most important, to the surgeon, of these agglutinative or serum reactions are those which are observed in typhoid fever and tropical dysentery, both of which, when the technical details are carried out with proper care, and when positive results are secured, are definitely instructive signs of great utility. On the other hand, they are nearly worthless when only negative results are obtained.

THE CEREBRO-SPINAL FLUID.

The technique of lumbar puncture is so simple, and the evidence furnished by analysis of the cerebro-spinal fluid often so valuable, that it should be constantly employed in the differential diagnosis of surgical diseases of the brain and cord. By the relief of intracranial and spinal pressure it also becomes occasionally a measure of considerable therapeutic value. Differential diagnosis between uræmia and meningitis, frequently so difficult, is occasionally rendered

EXPLANATION OF PLATE IV.

(Preparations Stained by Goldhorn's Polychrome Methylene Blue.)

Fig. 1.—Blood Smear from a Case of Trichinosis. The eosinophilic leucocytes are markedly increased in number, both relatively (14%) and absolutely. The red corpuscles show some diminution in hæmoglobin staining and a few microcytes are present, but the anæmia at this stage of the infection was not a marked feature of the case. The condition was accidentally discovered in the course of routine examinations of the blood in a surgical ward.

Fig. 2.—Pernicious Anæmia. The case was originally supposed to be one of gastric carcinoma.

The red corpuscles are few in number, but their hæmoglobin index is high above normal, a condition quite characteristic of pernicious anæmia. Marked poikilocytosis is present and microcytes and macrocytes exceed normal-sized red corpuscles in number. Polychromatophilic degeneration is marked in some of the cells, and both normoblasts and megaloblasts are present in considerable numbers. One of the latter cells shows karyokinetic changes in its nucleus. Megaloblasts are more or less diagnostic of pernicious anæmia, though also occasionally found in cachectic anæmias.

Fig. 3.—Polynuclear neutrophilic leucocytosis (32,000 Leucocytes per c.mm.), Occurring in an Acute Attack of Appendicitis in a Chlorotic girl.

The relatively high number of polynuclear leucocytes is well shown in this typical field. The chlorosis is indicated by the low hæmoglobin index, low red-cell count, and poikilocytosis.

Fig. 1.

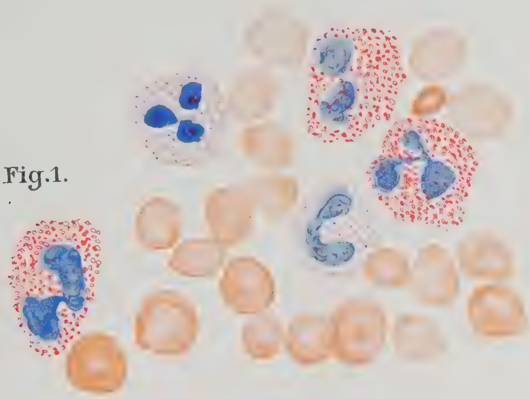
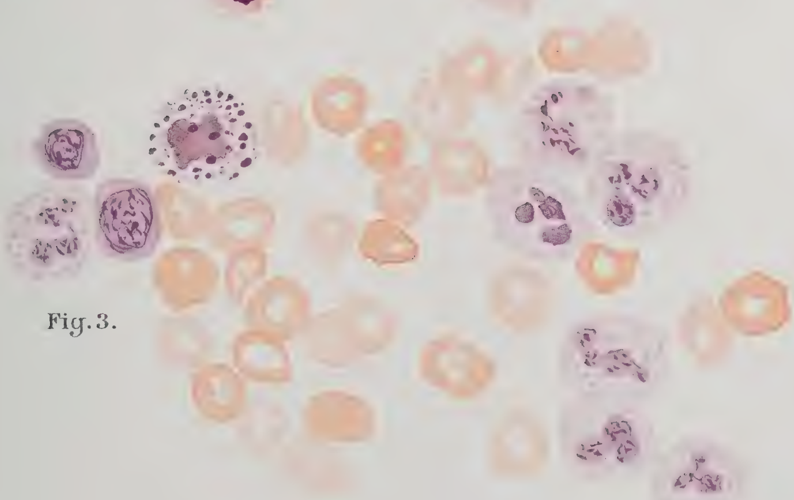


Fig. 2.

Fig. 3.



Harlow Brooks Del.

Lith. Anst. v. E. A. Funke, Leipzig.

Plate Illustrating Various Blood Lesions.

easy by this means. Ventricular hemorrhage may, in a certain number of cases, be differentiated from subdural hemorrhage by the fact that the fluid withdrawn is diffusely blood-stained, whereas in the latter condition the cerebro-spinal fluid is generally clear.

Inflammatory conditions of the meninges are indicated by a turbid, purulent fluid which clots readily, while the formed elements present are chiefly desquamated endothelial cells and polynuclear leucocytes or pus cells. In tuberculous disease, however, clot formation is usually but slightly marked, the fluid is more clear, and lymphocytes commonly predominate in its sediments. The cerebro-spinal fluid may also furnish most important aid in establishing the differential diagnosis between localized cerebral abscess or thrombosis and diffuse meningitis.

In certain specific inflammatory diseases of the meninges, as in epidemic cerebro-spinal meningitis or, more rarely, in tuberculous meningitis, it is possible to demonstrate, either by means of smear cultures or by animal inoculation, the specific cause of the disease,—frequently a matter of the greatest importance.

Although lumbar puncture is stated by most authors to be absolutely without danger, too rapid or too complete removal of the fluid may occasionally cause immediate collapse and death. Although surgeons need no warning as to the danger of infection of the cerebro-spinal space by careless methods, this possibility should always be held in mind.

THE SECRETIONS.

In our discussion of the different secretions we shall consider only those alterations which throw light upon certain diseased conditions of the body, or which modify these several fluids.

The Saliva.

An increase in the amount of the saliva may indicate mercurial, iodide, or other mineral poisoning, the specific kind of which can be easily determined by chemical methods. Decrease in the amount may also signify poisoning, as from atropine, or it may indicate that one or more of the salivary ducts is occluded, as by a calculus. Chemical examination of the saliva is frequently resorted to for the determination of the rate of gastric or intestinal absorption. One of the iodides or some one of the drugs which are excreted by the saliva is administered, and the time which elapses before it appears in this secretion measures quite accurately the rapidity of absorption. The determination of the digestive action is rarely necessary in surgery, but may be utilized in appropriate cases.

Tubercle bacilli, actinomyces, gonococci, diphtheria bacilli, and other specific micro-organisms may be found in the saliva, either as contaminations from diseased foci in the buccal cavity or as an evidence of a diseased state of the

salivary glands. Pus cells, blood, and desquamated epithelium may be discharged from the duct of an infected gland, and the finding of these products may furnish the first indication that any such disease exists.

The Gastric Juice.

An examination of the contents of the stomach is often of the greatest possible assistance in surgical diagnosis, and therefore it should never be omitted in cases in which some organic disease of that viscus is suspected. Where vomitus is not available, a test meal should be ordered, and after a definite time the contents of the stomach should be removed through the tube and submitted to both morphological and chemical analysis. In surgery and medicine alike probably the most important single investigation in connection with the gastric secretion is the determination of free hydrochloric acid. The diminution in quantity or the absence of this acid, when the condition is found to persist, is strongly suggestive of gastric carcinoma, particularly when deficiency in free hydrochloric acid is associated with the presence of lactic acid. The presence of free hydrochloric acid by no means excludes the diagnosis of carcinoma of the stomach, since it is often found present, even in normal amounts, particularly when the cardiac extremity of the stomach is largely intact. It is a noteworthy fact that free hydrochloric acid is rather more commonly found in sarcoma than in cancer of the stomach. The diagnosis of carcinoma of the stomach is further corroborated by the presence of broken-down blood in the fluid contents of this organ. Occasionally particles of new growth may be found in the fluid, and, on being submitted to microscopic examination, they may definitely establish the diagnosis. On the other hand, when blood is found associated with abundant free hydrochloric acid, this circumstance is considered indicative rather of ulceration than of a cancer. As a rule, lactic acid is absent in cases of simple ulceration; when it is present it is generally found in cases of pyloric ulcer, in which affection there is apt to be gastric dilatation with more or less fermentation of the food.

An interesting and often highly instructive test is that which consists in administering to the patient a measured amount of food or drink, and then, after a definite period of time has elapsed, withdrawing it for examination. In this way the peptic capabilities of the stomach and the degree of pyloric permeability may be ascertained. It is also an easy matter, in cases of dilatation, to determine, by actual measurement of the fluid contents of the stomach, just how far the distention of the organ has progressed.

The finding of certain forms of bacteria in the stomach may be of diagnostic import; nevertheless, too much reliance should not be placed upon such evidence, since the presence of bacteria is largely determined by the food. In my experience the occurrence of the Boaz-Oppler bacillus is of no value in the diagnosis of cancer of the stomach.

The discovery of pus in any considerable amount is much more suggestive of some extraneous suppuration draining into the stomach than of a suppurative gastritis. In these cases the pus should always be carefully studied, in the hope that it may contain cells or organisms which by their character indicate the origin and location of the primary suppuration.

Indol, skatol, bile, or even simple fecal odor may assist materially in the diagnosis of ileus, while parasites and their ova are by no means uncommonly found in the gastric contents, either indicating infection from the gut or parasitic disease of the stomach itself.

The Nasal Secretion.

Study of the nasal secretion is often of considerable value, particularly in infectious diseases, such as diphtheria, tuberculosis, or cerebro-spinal meningitis. The clinician must, however, remember that pathogenic bacteria, notably meningococci, diphtheria bacilli, and pneumococci, may exist in the nasal passages without disease necessarily taking place, although in these cases even slight traumatisms to the nasal mucosa may be followed by serious infection, such as probably takes place in the development of epidemic cerebro-spinal meningitis. When pus is found in any considerable amount, its point of origin should be determined. A good deal of pus may originate from inflammation of the mucosa only, in which case the exudate is very apt to be characterized by the presence of a good many eosinophile cells; but large amounts of pus are much more likely to be due to suppuration of the antrum.

Where nasal growths exist the number of epithelial cells in the secretion may be considerably increased, but no more so than in some simple catarrhal processes.

Investigation of the nasal secretion is necessary in traumatic injuries of the head; for example, in fracture of the base of the skull, when cerebro-spinal fluid may be found escaping from the nose. This fluid may be recognized by its non-albuminous character and by the presence of a substance which reduces Fehling's solution. In a case of recent fracture, the specimen of fluid collected is commonly more or less stained with blood, but in certain instances of cerebral tumor, and also in some cases of hydrocephalus, cerebro-spinal fluid may be found escaping from the nose, often in such quantities as to cause great annoyance to the patient. In cases of this nature the fluid is, of course, ordinarily free from blood, except such as may enter from the nasal mucosa, which is also likely to contribute the mucus, leucocytes, and desquamated epithelium normal to the nasal secretion.

The Sputum.

The sputum, which normally consists chiefly of the secretions of the tracheal and bronchial glands, often well repays study, though to a much less degree in surgery than in internal medicine.

Tubercle bacilli, pneumococci, actinomyces, and other specific organisms in the sputum are of great significance, while cells and particles of tissue from pulmonary tumors may be discharged in the same manner, thus materially assisting diagnosis. In nearly all cases of inflammatory disease of the respiratory tract, blood cells, fibrin, leucocytes, and pus are present to a greater or less degree, and are often indicative of the character, extent, and standing of the focus

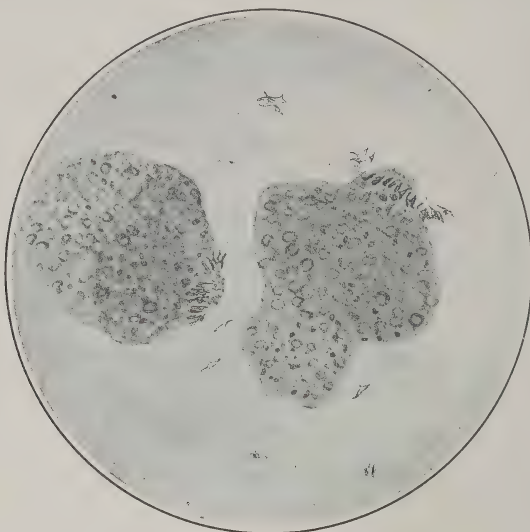


Fig. 143.—Echinococcus. Embryos and hooklets discharged in the sputum from a case of hydatid cyst of the lung.

from which they arise. Pus, sometimes mixed with hepatic cells, may be discharged through the sputum from a liver abscess, which not uncommonly bursts into the lung; in the same manner echinococcus hooklets, nodules of actinomycotic material, etc., may be discharged (Fig. 143).

In gangrene of the lung or in extensive tuberculosis considerable masses of pulmonary tissue may be expectorated, and may be of such character as to demand microscopic examination for their definite recognition.

The Mammary Secretion.

In most surgical diseases affecting the mammary gland when functionally inactive, examination of the secretion, as a rule, shows little of direct value. On account of the superficial location of the gland, which renders its physical examination relatively easy, studies of the secretion are not generally necessary. Nevertheless, at times they become highly valuable. Sufficient secretion may, in certain conditions, be expressed from the nipple or gland to give on examination a fairly certain knowledge of the changes which are taking place. Blood or pus may indicate the formation of an abscess, and the bacteria found in the secretion may show its cause. In the by no means rare cases of mammary tuberculosis, tubercle bacilli are not, as a rule, demonstrable in the secretion. In

certain tumors of the breast, particularly adenomata, cells and secretions more or less characteristic of the growth may be discharged.

Examination of the secretion during lactation is rarely necessary in surgery; at most, it may be thought desirable in a case in which suppuration or a neoplasm is suspected. The variation in the quantity or quality of the milk in surgical disease may, however, become of importance, since in so many conditions, such as shock, the amount becomes diminished or the character changed.

Secretions of the Female Genital Tract.

In many conditions affecting the female genital tract, and particularly in the infectious diseases which so commonly affect these parts, systematic study of the secretions becomes a matter of necessity. Not only should all abnormal discharges receive attention, but the apparently normal secretion of each glandular distribution should be investigated. Thus, gonorrhœal affection of the vulvo-vaginal glands may exist without vaginal infection and vaginal infection without cervical involvement. The results attending these examinations may be somewhat confusing, unless one bears thoroughly in mind the character and great variation of the secretions normal to these parts. Thus, the vaginal secretion, in addition to leucocytes and desquamated epithelium normally derived from its own wall, is almost invariably more or less composed of materials from the endometrium of the uterus and cervix. Just before, during, and for a considerable time after menstruation, blood, broken-down epithelium, leucocytes, and frequently more or less pus are present in the discharge; but such material found unassociated with menstruation would be strongly suggestive of endometritis or even of a new-growth of the uterus.

Similarly, the desquamated epithelium naturally present in the vaginal secretion may be confused with products due to the erosion of a tumor. In every case of purulent discharge from the genital tract the material should be especially examined for the bacteria present. Gonococci, tubercle bacilli, and members of the proteus group of bacteria can, as a general rule, be satisfactorily recognized—at least so far as it is necessary to do so for clinical purposes—by the examination of smears alone. Wherever it is deemed of great import that the precise character of any inflammatory process be decided, bacterial cultures should be made; this is often of great value, particularly in post-partum infections. Before a final negative conclusion is reached in any case of infection, several examinations should be made.

Occasionally there are found, in the matter discharged from the uterus, portions of tissue, the examination of which may definitely decide the important question between some form of tumor of the endometrium and the products of inflammation or conception. I have found it in all cases much the safest procedure not to rely on gross or direct microscopic examination only of this fresh material, but to prepare and examine it after the usual histological methods.

Unless this course be adopted as a rule, serious diagnostic mistakes will inevitably occur.

An examination of the vaginal secretion sometimes reveals the presence of parasites or their ova, of which the thread-worm and the Trichomonas vaginalis are probably the ones most frequently met with. It is needless to say that an absolute diagnosis of disease of the genital tract of the female should never be made on the examination of the secretions alone; this should be but supplementary to as complete a physical examination as is practicable in any particular case. Where growths or swellings suggestive of syphilitic infection are present, the secretions from them, or, if necessary, expressed blood or serum, should be searched for the Spirochæta pallida, since it now appears that this organism is quite constantly associated with recent syphilitic lesions.

Secretions of the Male Genital Tract.

In diseases of the genito-urinary tract of the male the urethral secretions should be investigated; early examination frequently establishes the diagnosis, for example, of a gonorrhœal urethritis, before the clinical symptoms have developed. Streptococcus, pneumococcus, and other infections may be detected in the same manner, as may also tuberculous disease. As regards the tubercle bacillus the differentiation from the other acid-fast bacilli should be made certain, if necessary, by animal inoculation.

Where prostatic disease is in question, the secretion from the prostate may be readily obtained by first causing the patient to urinate or by otherwise washing out the urethra, after which massage of the prostate and seminal vesicles drives the secretions from these glands into the urethra, from which locality they may be secured for examination. Gonorrhœal, tuberculous, and other infectious processes are readily detected in this manner. In cases in which syphilitic infection is suspected, a search for Spirochætæ pallidæ should be made.

When lesions of the testis or epididymis are present, much may be learned by an examination of the seminal secretion. Pus and blood in more than minute amounts may indicate inflammatory disease; or blood alone, a new-growth. The demonstration of bacteria in the secretion may absolutely decide the nature of the process, and the presence or absence, character, and motility of the spermatozoa may throw much light on the true character of the lesion. The permeability of the epididymis and of the vas deferens may also be determined by such an examination of the secretion in the urethra, and the surgeon is now and then surprised at the considerable number of cases in which few or no spermatozoa reach the urethra. A discovery of this nature serves, in not a few cases, to determine the proper course of surgical treatment. Surgeons are too apt to neglect this very simple and often decisive method of examination, even in those cases in which it is perfectly practicable to secure the secretion for investigation.

Other Secretions.

In the various bacterial diseases of the eye the lachrymal secretion may furnish evidence of the specific type of infection of the conjunctiva or of its adjacent glands and mucous tracts. In exploratory procedures it is occasionally possible to secure the secretions from the internal viscera, as that of the pancreas or liver, and thus the surgeon may obtain direct evidence of changes taking place in these organs. In short, all of the secretions may, under diseased conditions, contribute facts which have an important bearing upon surgical diagnosis, not only in cases of local disease, but also in those of a more general nature.

THE EXCRETIONS.

The Urine.

Examination of the urine plays a large part in the diagnosis and study of nearly all disorders of the body. Its importance, however, is greater in the domain of internal medicine than in that of surgery. Its chief value, in the latter domain, is to be found in the special surgery of the urinary organs.

One point of great importance in connection with the examination of the urine—a point which has but recently received adequate attention—is that the amount and character of the food, drink, and medication have a most direct and important bearing on the urinary picture. An examination of the urine should, therefore, be prefaced by a close inquiry into this important question, so that the urinary findings may not be incorrectly interpreted. Thus, for example, the amount of water taken as food or drink, and the quantity excreted by the skin, respiration, and bowel determine to a large extent the color, reaction, and specific gravity of the urine. Articles of food, such as rhubarb and asparagus, and even excessive amounts of albumin or sugars, or drugs, such as phloridzin, may cause marked and apparently serious alterations in the color, odor, and chemical nature of the urine.

Another frequent source of error lies in the manner in which the specimen is collected, and it should always be borne in mind that the urine may be contaminated by substances derived from the bladder, prostate, and urethra; or by materials entering the urine from the external genitals, the rectum, or the vagina in women; from the air, or from the vessel into which the specimen is voided. Substances found in the urine, particularly when of unusual nature, should never be considered as of clinical significance until all these possibilities of contamination, which may even be wilfully effected, have been considered and excluded.

Ureteral catheterization has furnished us with the means of determining accurately the condition of each one of the kidneys separately, and, since it has been shown that under normal conditions both organs excrete alike, we can now

definitely decide whether one or both are involved in any disease process, and to a certain extent we may also conclude as to the relative extent of the disease. This precaution is of particular bearing on cases in which operative measures on one or both kidneys are contemplated.

As already intimated, the amount, color, reaction, and specific gravity of the urine are to be considered in connection with the amount of fluid ingested as well as with that excreted elsewhere. When these do not appear to be normally balanced, there is reason to suspect the existence of disease. The precise nature of the disorder must be determined by further investigation, which demands examination of the urinary excretion for the entire twenty-four hours, after the patient has been placed upon a simple, though normal and easily determined diet, as of measured amounts of bread, milk, and lean meat.

Much more valuable data for the estimation of these points is to be derived from kryoscopy of the urine, by means of which the molecular concentration may be determined from its freezing-point. For the proper understanding of this, the same method must also be applied to the blood and to any transudates which may be present in the case. Any discrepancy between the normal balance—as, for example, increased concentration of the blood with decrease in the molecular concentration of the urine—indicates deficient renal activity.

A procedure presenting fewer technical difficulties to the surgeon, and at the same time giving an accurate test of the permeability of the kidney, is afforded by the administration of phloridzin or methylene-blue and the determination of the time required for its easily recognized appearance in the urine. Where ureteral catheterization is also practised, the relative activity of the two kidneys may thus be ascertained.

The amount of urea excreted in the urine is rarely of much importance to the surgeon when the rate of permeability of the kidney has already been demonstrated; but when the amount remains high in the presence of a light nitrogenous diet, it is strongly suggestive of tissue destruction, as in diabetes. Decreasing excretion of urea is shown in such conditions as Weil's disease, acute yellow atrophy of the liver, and in some cases of diffuse carcinosis. The respective amounts of urea and uric acid excreted in surgical conditions are of relatively great importance in surgery, on account of the diminished solubility of uric acid and the consequent tendency to precipitation and calculus formation, either when it is thrown out of solution in the kidney on account of increased excretion, or when the chemical characteristics of the urine are such as favor its precipitation.

Albuminuria is of considerably less significance in surgery than in medicine, except for that which takes place in hæmaturia or in post-operative cases. Albuminuria may occur in surgical shock, after operations, particularly where extensive manipulation of the tissues has been necessary, after injuries to the head, in carcinomatous peritonitis, in abscess of the liver, and in nearly all the diseases

of an infectious nature. It may follow the administration of many drugs, notably those of an irritant nature, or may occur after the observance of a certain diet—for example, the ingestion of egg albumin. In certain cases it may also appear apparently as an individual peculiarity without demonstrable disease or other cause. The occurrence of albumin in the urine in surgical disease is of great importance only when its causation is of a serious surgical nature, and albuminuria *per se* is no longer of the grave significance which the text-books of a few years ago led us to believe. It may occur apparently without any connection with renal lesions, and, conversely, kidney disease, often of the most grave nature, may exist without albuminuria. The simple presence of albumin in the urine, where renal permeability is within the range of normal, is now no longer considered as contraindicating operation or the administration of a general anæsthetic, though its cause be distinct renal disease. Extra percautions in the selection and use of the anæsthetic may, however, be necessary.*

When albuminuria occurs as a manifestation of the escape of blood into the urine its surgical importance is great, and the probable point of entrance of the blood must be ascertained, as well as the cause. Such an escape of blood may occur from a renal or cystic neoplasm, from a stone located in the kidney itself, in the ureter, or in the urinary bladder; from some inflammatory condition or from a simple congestion of the kidney; or, finally, from some traumatism of the bladder or urethra.

Glycosuria is also of less interest in surgery than in general medicine, though its occurrence in surgical diseases is often of the most grave significance, both from the standpoint of diagnosis and from that of surgical therapeutics. The wide difference between simple glycosuria and glycosuria as a symptom of diabetes mellitus must be fully appreciated, since the former condition may be but a temporary one and due perhaps to individual peculiarities, dietetic conditions, or the use of certain drugs, as phloridzin; while the latter is a disease in which glycosuria is a single manifestation and in which gangrene, delayed healing, and fatal coma are notoriously prone to occur. Temporary glycosuria may take place in injury to the head or in certain cases of mental or physical shock.

Indican is a product of albuminous decomposition which occurs in the urine in the case of excessive putrefaction, particularly in the stomach and small intestine. Simon asserts that its presence and amount are of diagnostic importance, since it is found particularly in cases of derangement of the gastric secretion or of the motor powers of the small intestine. Its appearance, in my opinion, is too inconstant and indefinite to render it of any great diagnostic value.

The occurrence of bile pigment in the urine takes place when there is any obstruction to prevent the normal flow of the bile into the intestine, as may oc-

* The special chemical nature of the albumin excreted is often more or less diagnostic as to its cause; thus Bence-Jones albumin, which is easily recognized by its special reactions, occurs in most if not all cases of multiple myeloma.

cur from an impacted gall stone, from catarrhal swelling of the mucosa of the duct, or from the pressure of a neighboring tumor or an inflammatory deposit. It also takes place whenever from any cause the liver is unable to convert the waste blood pigment normally, as in acute yellow atrophy or in Weil's disease, or when an excessive destruction of the corpuscular elements of the blood throws an overabundant supply of pigment on the liver, as in malaria, pernicious anæmia, and like maladies. Bile in the urine is almost always associated with more or less jaundice, so that its diagnostic value is ordinarily only corroborative and necessitates a much wider investigation for the determination of its cause.

The estimation of the chlorides of the urine is not of much value in surgery, except as a means of showing the degree of absorption from the gastro-intestinal canal. They are decreased, sometimes very markedly, in many acute infectious diseases, notably in acute inflammation of the lung.

The phosphates of the urine are also of relatively little importance, except as they may be deposited in the renal pelvis or bladder, and so tend to the formation of stone. They are much augmented in extensive tissue destruction.

Unquestionably the most valuable facts derived from the examination of urine in surgery are those secured by microscopical study. In this relation care must always be taken not to interpret incorrectly certain bodies which normally occur in the urine; thus, epithelial cells in greater or less numbers are naturally eroded from the mucosa of the kidney pelvis, from the bladder and urethra, and from the skin and mucous membrane of the external genitals. Further, when epithelial cells in small numbers only are found, absolutely nothing definite can be told from their morphology as to their point of origin—a fact of simple elemental knowledge of normal histology which is too often forgotten by over-hopeful and inexperienced microscopists. When large flakes of epithelium are thrown off, a certain amount of probability may be given to statements as to the point of origin, but nothing of sufficiently definite character to warrant the adoption, on this basis alone, of operative measures. Occasionally considerable bits of tissue may be thrown out into the urine, particularly in necrotic forms of inflammation or in papillomatous neoplasms.

Too much reliance must not be placed on the presence or absence of casts. Hyaline casts, even in considerable numbers, may be present in the urine from apparently normal kidneys, particularly after diuresis, and even granular casts in small numbers may be found with a relatively normal excretion; conversely, extensive and even fatal nephritis or uræmia may exist without the presence of casts. Blood, epithelial and pus casts are, of course, indicative of disease; and, as a rule, granular casts in any considerable number are of similar import.

Blood in the urine, if certainly derived from the urinary tract, is a most important surgical sign. When hæmoglobin, with perhaps blood serum only, is found, as in the hæmoglobinuria of profound malaria or in toxæmias associated with extensive destruction of blood corpuscles, or in such states as Raynaud's

disease, no lesion of the kidney itself is indicated. When red corpuscles are found in the urine, it is permissible to assume that in some part of the urinary tract there exists profound congestion or even inflammation, or that there is a new-growth or a granuloma. The quantity of the blood and the time when it appears in the urine, taken with the other clinical symptoms and signs, usually indicates sufficiently where the lesion is located.

Pus in the urine is, of course, confirmatory of inflammatory disease in the urinary tract. When it is continuously well mixed with the urine, suppuration of the renal tissue or pelvis is indicated; when it is associated with blood and crystals, stone is to be considered; or when, in addition to the pus, there are fragments of tissue and blood, a neoplasm or tuberculosis is suggested. Wherever pus is found in the urine, specimens secured under aseptic conditions should be examined bacteriologically. Occasionally mere examination of properly prepared smears is sufficient, as in gonorrhœal infection; but often bacterial cultures, with isolation of all the organisms present, are necessary. Where tuberculosis is suspected it is well not to rely exclusively on a smear examination in any case, both on account of the great rarity of the organisms in some instances, and on account of the difficulty often presented in the distinction of the tubercle bacillus from other acid-fast bacteria frequently present in the urine. In these cases animal inoculation is the only safe procedure.

Crystalline and amorphous deposits in the urinary sediment are of great surgical significance, particularly when renal or cystic calculus is suspected. The most important of these deposits are uric acid or urates, calcium oxalate, and triple phosphate. Amorphous phosphates, when out of solution while the urine is still in the body, are also frequently involved in the formation of stone, particularly when associated with various gums and colloidal bodies which are actively concerned in the precipitation of urinary salts and in the formation of stone. Rare crystals of xanthin, leucin, or tyrosin, and infecting protozoa, as the trichomonas or Balantidium coli, are infrequently of much surgical interest.

The Fæces.

Examination of the fæces furnishes a most direct means of ascertaining not only the condition of the digestion and the absorptive powers of the gastro-intestinal tract, but also, in many cases, the state of outlying viscera and the presence or absence of general as well as local disease. As with the examination of the urine, account must be taken of the food and drink before inferences are drawn from the appearance or from an analysis of the stools. Thus, a largely vegetable diet gives rise to greater and softer movements than one mostly of meat; foods rich in chlorophyl give a green color; milk gives rise to abundant light yellow movements; while drugs, as iron, bismuth, manganese, as well as certain berries, give a dark, almost black color.

Certain inferences are to be drawn from the shape of the fæces. Narrow,

ribbon-like excreta are formed in rectal stricture or in nervous spasm of the anus; small, round, scybalous masses occur in constipation or sometimes as a result of a highly nitrogenous diet.

Gross inspection often suffices for the detection of such bodies as the larger parasites (Fig. 144), round worms, segments of tænia, and seeds or pits of fruit, bits of undigested vegetable matter, gall stones, and the like. Ordinarily steatorrhœa can also be diagnosed by gross examination; the light clay color of the movement, with contained flakes or globules of white fat, indicating deficient bile flow, disease of the pancreas, or perhaps a diet over-rich in fat.

Blood in the stools may be readily discovered in many cases by the unaided eye. When it is found fresh and bright red in color, particularly streaking the

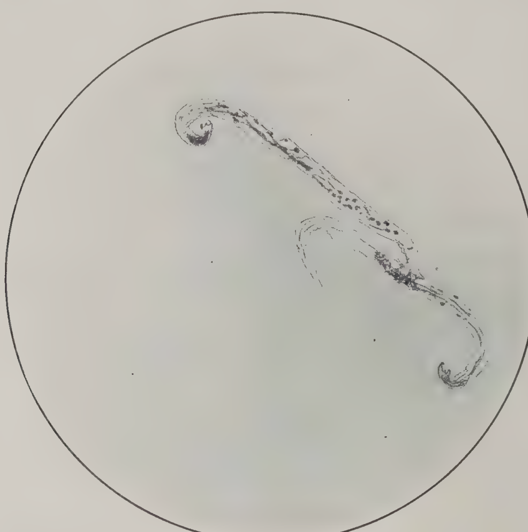


Fig. 144.—Uncinaria americana, Male and Female. From a case of severe anæmia occurring in a soldier returned from Porto Rico.

surface, hemorrhage from the anus or rectum, as from hemorrhoids, is suggested. Large amounts of fluid or clotted blood, but slightly altered, indicate hemorrhage, perhaps from ulceration in the lower portion of the small intestine or from the colon. When the blood is well mixed with the stool and more or less digested, the color of the movement becomes black or dark green and cannot be definitely diagnosed without the use of the microscope or by chemical means. Such findings are observed in intestinal or gastric hemorrhage, where the amount of blood lost is not large, and they may signify small ulcers, or cancer of the stomach or small intestine, or perhaps simple congestion, as might take place in cardiac incompensation or portal thrombosis. Intestinal hemorrhage, which, if known, would be of the greatest diagnostic value, may take place without producing in the stool alterations sufficient to attract the naked eye or to be discoverable upon microscopic examination. Thus, in the early stages of cancer of the stomach, in hook-worm infection, in cirrhosis of the liver, and in many

other conditions in which only minute quantities of blood escape into the bowel, chemical examination of the stool may be necessary, and the detection of blood by the guaiac or other chemical tests becomes in such instances of great value.

Mucus in excessive amounts ordinarily indicates catarrhal colitis, and, when it is streaked or flecked with blood, ulceration is to be considered, in which case bits of eroded tissue, globules of pus cells, and small masses of fibrin are ordinarily present. When considerable masses of tissue are passed the surgeon is justified in suspecting the presence of a new-growth, and, in some cases, portions sufficiently large to warrant histological examination may be secured and may render an absolute diagnosis of cancer or papilloma possible. It must not be forgotten, however, that animal tissue from the food occasionally passes through the entire intestinal tract with but little change, and it is therefore wise to consider animal tissues as always derived from the food until it can be shown that they emanated from another source. Epithelial cells desquamated from new-growths are rarely in sufficient number to excite suspicion, and this is due to the simple fact that the cells which are eroded from the intestinal mucosa, under natural circumstances or as the result of a simple inflammation, are very numerous.

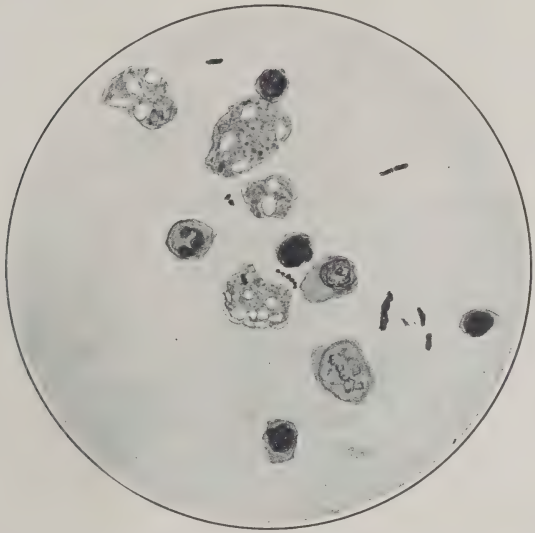


Fig. 145.—Amœbæ coli. From the fæces of a case of tropical abscess of the liver occurring in a soldier recently returned from the Philippine Islands.

In all the departments of medical science study of the fæces for the detection of animal parasites is demanded, and in no branch more so than in surgery. The discovery of ova of the anchylostoma, for example, satisfactorily explains the type of often actively progressive cachectic anæmia which is more than occasionally mistaken for that of a malignant tumor. Demonstration of amœba coli (Fig. 145) in the fecal discharges frequently clears the diagnosis of abscess of the liver, while the occurrence of ova or segments from the various tæniæ or round-

worms often explains otherwise confusing symptoms. Bacterial examination of the fæces yields in some cases valuable surgical data. The mere demonstration of typhoid or dysentery bacilli, now no longer a matter of great technical difficulty, renders the diagnosis of these diseases at once final and conclusive.

Perhaps, however, no more valuable facts are ascertained for the surgeon by the examination of the fæces than are given him by careful daily study in convalescent cases, particularly after laparotomy—cases in which, by the aid of such regular examinations, the diet may be regulated according to the digestive and absorptive peculiarities of each individual patient.

THE TRANSUDATES.

The importance of the examination of the transudates lies in the fact that we are thereby enabled to differentiate them from the exudates. The transudates are of passive origin, occurring in hydræmia, in circulatory disorders, and in affections in which the liquids are insufficiently excreted, owing to defective action on the part of the kidneys, skin, or bowel. Any of these conditions, therefore, may be suggested by the presence of transudates.

The character of transudates differs somewhat, both chemically and morphologically, according to their location. Thus, those from the pleural cavities are ordinarily of somewhat higher specific gravity than those occurring elsewhere.

Sedimentation and microscopic examination generally disclose a few epithelial cells, macerated and desquamated from the walls of the involved space, or blood cells, usually very much distorted or swollen.

Absence of clot, low percentage of hæmoglobin, and lower specific gravity distinguish the transudates from the exudates, which may be further differentiated when necessary by kryoscopy.

THE EXUDATES.

Since the origin and nature of the inflammatory exudates are fully discussed in an earlier chapter, we shall consider them here briefly and only in their immediate bearing on surgical diagnosis.

For the purposes of surgical diagnosis systematic examination must be practised wherever exudates are found. An examination of both the gross and the microscopic appearances, and also a bacteriological investigation, are of much more importance than chemical methods, although these must also be occasionally employed.

The exudates may be serous, hemorrhagic, chylous, chyloid, putrid, or purulent. Their coagulability and general character can, as a rule, be determined with the unaided eye at the operating table, but the microscope is usually necessary to furnish the more important data as to probable origin or precise type.

From the character of the cells contained in the exudate its etiology can often be accurately determined.

Serous exudates closely resemble transudates. After they have stood for a short time, however, a moderate amount of clot usually separates out and sedimentation shows the presence of formed elements having more or less marked characteristics. Cells dislodged from the wall of the cavity into which exudation has taken place are usually demonstrable, and leucocytes, commonly polynuclear neutrophiles, are present in greater or less number. Where the exudate has been of long standing the cells usually show hydropic degeneration, and may eventually be represented only by amorphous detritus.

Hemorrhagic exudates are characterized by the presence of blood in considerable amount, for nearly all serous exudates and even the transudates contain a few red blood cells. They may occur after traumatism, in pernicious anæmia, purpura, hæmophilia, and in similar hæmic diseases, but are most commonly seen associated with tuberculosis or malignant neoplasms.

Where the transudate is tuberculous in character, it is usually difficult to demonstrate the tubercle bacillus except by animal inoculation. In the case of cells derived from a new growth, it will often be found that they exhibit karyokinesis. Furthermore, such cells are sometimes present in such numbers as to give the fluid a milky turbidity. When small pieces of the new-growth are found in the fluid, then it is sometimes possible to make an absolute diagnosis from an examination of the exudate alone.

Chylous exudates are met with in cases in which lymph channels of considerable size have broken into the body cavities. They are rare, but are readily recognized by the presence of fat and oil globules, which are suspended in the fluid in such a manner as to give it the appearance of milk, both macroscopically and microscopically.

Chyloid exudates occur when extensive destruction of epithelial cells takes place, as a result of which destruction minute fat globules and much cell detritus are set free. Such exudates are mostly found in cavities the walls of which are either cancerous or tuberculous.

Putrid exudates are recognized by their foul odor; they are usually dark in color, and their sediment is made up of necrotic detritus only.

The purulent exudates contain pus, and their chief characteristics are those of pus. In certain conditions the gross appearance alone of the pus suffices to reveal its etiology or character. Thus, in suppuration caused by the bacillus pyocyaneus the exudate is green in color, and infection with the yellow staphylococcus is often productive of a pus having a deep golden hue. When mixed with blood, the fluid acquires a characteristic bloody tinge; when it emanates from a tuberculous focus, it is very apt to contain curds and coagula of whitish-gray necrotic material. The characteristics of pus in the various processes are in the main determined by the etiological factors concerned in its production.

For this reason particular attention should be paid, in the examination of pus, to the discovery of its etiology. In many cases this may be possible from the simple examination of smear preparations, as in tuberculous, gonorrhœal, and diphtheritic exudates. In all cases special staining methods should be employed, viz., such as are calculated to bring out the factors supposed to be present in each case; or, when necessary, several methods should be used, as are, for example, sometimes required for the absolute identification of the gonococcus.

Where many bacteria of different sorts are present, it is usually impossible, except in the case of such specific inflammations as tuberculosis or gonorrhœa, to decide, from this brief examination, which is the more important. In these cases, as well as in those in which no bacteria or protozoa can be demonstrated in smear preparations, cultures on appropriate media, and often under both aërobic and anaërobic conditions, must be made, and the various organisms isolated, and their respective virulence tested, if necessary, by animal experiment. In some cases direct animal inoculation is to be preferred, particularly when the question of tuberculous infection arises and when the bacilli are not sufficiently abundant to admit of easy detection by the ordinary staining methods.

Certain specimens of pus are best examined fresh, as, for example, the pus of liver abscess, for the reason that the demonstration of the amœba coli is more easily accomplished in this manner than when it has been stained; but, as a rule, stained preparations are to be preferred.

Pus should always be carefully searched for cells or bits of tissue that may have been dislodged from the primary seat of the disease, which may be thus disclosed. This is often of value, particularly in malignant tumors, where active growth is closely associated with necrosis.

Certain points as regards the age of pus may be determined by submitting it to a microscopic examination. Thus, when it is of rather recent formation, the leucocytes and other cells contained in the fluid are as a rule well preserved, and the pus cells retain to a large extent their typical neutrophilic granule-staining reaction. On the other hand, when the pus is old these bodies are largely or entirely broken down, the serum may have become absorbed, and the pus may be represented only by a sterile, cheesy material, sometimes more or less calcified.

CYST CONTENTS.

Diagnosis as to the origin of most cysts is possible from the examination of their contents. In many cases such an examination is of the utmost importance to the diagnostician, particularly when differentiation between true cysts and neoplasms which have undergone cystic degeneration is necessary. Encapsulated accumulations of inflammatory exudates, resembling cysts, may also be recognized in this manner. In some cases microscopic study of the material aspirated from the cavity suffices for the diagnosis, as in the ordinary ovarian cysts, but in a certain number of cases chemical investigation is also necessary.

Cysts of the kidney or simple hydronephrosis may be recognized by the detection of urea or uric acid in the cystic fluid. Hydatid cysts are manifested by the presence of the hydatid hooklets or scolices (Fig. 143), cysts of the liver by the presence in the fluid of bile-coloring matter, and pancreatic cysts may sometimes be identified by tryptic reactions obtainable with the fluid aspirated from them.

In the case of dermoids and the more solid cysts, as of the thyroid gland, diagnosis is commonly possible by the gross or microscopic inspection of the material removed, although aspiration of these cysts is often impossible, especially where hair or teeth-like structures are present. In nearly all instances removal of a portion of the contents through small incisions and the use of the microscope render diagnosis easy.

THE EPIPHYSES AND THEIR RADIOGRAPHIC INTERPRETATION.

By PRESTON M. HICKEY, M.D., Detroit, Michigan.

THE introduction of the Roentgen ray as a diagnostic aid in surgery has led to the establishment of a special field of study which may be termed radiographic anatomy. The intelligent use of the radiograph presupposes on the part of the observer some knowledge of the radiographic art, as well as some acquaintance with the normal and pathologic appearances. The successful use of the microscope as an aid in clinical medicine demands a preliminary laboratory training the results of which are valuable in proportion as the technique is exact and the observer experienced. To realize the value of radiography the proper construction must be placed upon the findings of the photographic plate. The lights and shadows coaxed forth from the creamy surface of the sensitive film by the chemical developer must be interpreted in the light of a previous training in this special field, and not judged through preconceived ideas based on inadequate data.

The radiographic study of the human bony framework during its process of development presents varying pictures which often prove deceptive to the inexperienced. Before taking up in a series the various plates which are obtained at different ages, it would be well to remember that all radiographs are produced according to the laws of projection and should be interpreted with a full understanding of these laws. The Roentgen rays given off from a Crookes tube properly energized proceed principally from a central point on the target of the tube. These rays diverge in a definite ratio.

Fig. 146 illustrates this point. The further removed the objects are from the photographic plate and the nearer they are to the target from which the rays emanate the greater will be their apparent magnification. Rays which come off at somewhat of a tangent to the target will produce more distortion than the more direct rays. The practical lesson to be drawn from these observations is that the target of the tube should be placed as exactly as possible over the part which is to be radiographed, and the part which we most desire to be clearly shown should be approximated to the photographic plate.

Bodies between an x-ray tube and the photographic plate cast shadows upon the plate proportionately to their atomic weights. In the human body, tissues containing lime cut off the ray more than does muscle or cartilage; hence,

the unossified ends of the bone cast such feeble shadows that they are practically invisible in the ordinary radiograph.

In Fig. 147 is presented a radiograph of a new-born child delivered at seven months; it shows at a glance the condition of the long bones and the wide spaces between them occupied as yet by only soft tissues. The striking flexibility of the joints and their natural protection against fractures at this early age are well illustrated. The epiphyses of the long bones, being still cartilaginous, are photographically invisible. The carpal and tarsal bones, with the exception of the os calcis, are absent.

The development of the carpus, from a radiographic standpoint, shows that the os magnum and the unciform are first noted in the order of appearance of

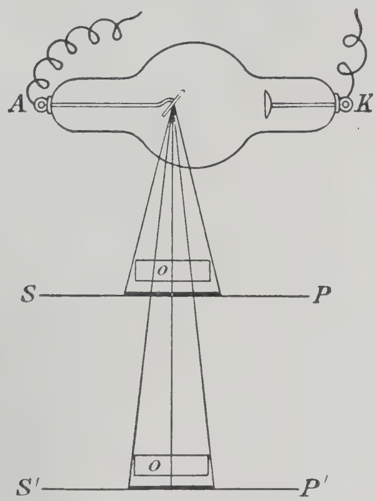


FIG. 146.—The Rays from the Target of the x-ray Tube, AK, diverge from one point so that the object O, met first by the rays, would appear larger on the photographic plate, SP, than it would on a similar plate (S' P') situated farther away from the tube.

the various osseous centres. These bones usually ossify during the first few months of life. In an infant at the age of fifteen months we are accustomed to find the lower epiphysis of the radius manifesting itself first as a small point. During the second year of life these two bones, the os magnum and the unciform, and the lower epiphysis of the radius increase in size, while as their growth advances we find that the proximal epiphyses of the first row of the phalanges begin to make their appearance.

The next carpal bone that can be distinguished is the cuneiform. Fig. 148, which represents the hand and wrist of a child at five years of age, shows four of the carpal bones present, namely, the os magnum, the unciform, the cuneiform, and the semilunar. The lower epiphysis of the radius is well formed. The lower epiphysis of the ulna has not yet appeared. The epiphyses of the phalanged bones are all more or less distinctly visible, while the metacarpals show their distal epiphyses.

Fig. 147.—Radiograph of a New-born Child Delivered at Seven Months. The epiphyses of the metacarpals and phalangeal bones have not yet appeared. The carpus is still cartilaginous and hence produces no shadows on the photographic plate. The humerus, radius, and ulna present no bony epiphyses. The lateral centres for the sacrum are distinct. The ilia are distinct from the os pubis to the ischia, which are united at their superior ends. The head of the femur produces no shadow, while in the knee joint the distance between the femur and the bones of the leg is quite striking. (Original.)

In Fig. 149, which represents the hand and wrist at eight years, we note the lower epiphysis of the ulna. The scaphoid, trapezium, and trapezoid also are distinct. The space between the ossified portions of these carpal bones is still considerable.

The lower epiphysis of the radius appears at a much later time than has been assigned by the older anatomists, usually at the age of about seven years.

In the wrist at eleven years (see Fig. 150) we find a proximal epiphysis—the epiphysis of a second metacarpal. The appearance of the epiphyseal line might easily be mistaken for a fracture line. The second metacarpal is peculiar in the fact that it usually develops from three centres.

FIG. 148.—Radiograph of Hand at Five Years. 1, Shaft of fifth phalangeal; 2, epiphysis of same; 3, epiphysis of fifth metacarpal; 4, os magnum; 5, unciform; 6, cuneiform; 7, semilunar; 8, ulna; 9, epiphysis of radius; 10, radius. (Original.)

In Fig. 151, which illustrates the wrist at twelve years of age, we note the peculiar notch at the proximal end of the second metacarpal, which marks the incomplete union of this bone with its proximal epiphysis.

In Fig. 152 we note the superimposed shadow of the pisiform as covered by the greater shadow of the cuneiform.

In considering the development of the carpus, from a radiographic standpoint, we must remember that there are considerable variations in the times at which the different wrist bones make their appearance. While the above statements will be true of the great majority of cases, there will, however, be some to which these statements do not apply. The irregularity may be present symmetrically in both the left and the right joints, although not infrequently

children are encountered in whom the osseous development of one carpus is strikingly different from that of its fellow on the opposite side. Local conditions—such, for example, as a pre-existing tuberculosis—may sometimes be the cause of this irregularity. None of these peculiarities, however, will give rise to any considerable difficulty in their interpretation.

Of all joints in the body, the elbow is the most interesting from a radiographic standpoint. This is due to the fact that there are so many different

Fig. 149.—Hand at Eight Years. 1, Ungual phalanx of thumb; 2, epiphysis of same; 3, proximal phalanx of thumb; 4, epiphysis of same; 5, first metacarpal; 6, epiphysis of same; 7, trapezoid; 8, trapezium; 9, os magnum; 10, scaphoid; 11, lower epiphysis of radius; 12, radius; 13, fifth ungual phalanx; 14, epiphysis of same; 15, second phalanx of little finger; 16, epiphysis of same; 17, third phalanx of little finger; 18, epiphysis of same; 19, epiphysis of fifth metacarpal; 20, fifth metacarpal; 21, unciform; 22, cuneiform; 23, semilunar; 24, lower epiphysis of ulna; 25, ulna. (Original.)

centres of development in the elbow, in consequence of which the appearance of the elbow joint varies radiographically year by year from birth to the age of sixteen. When radiography was first employed in the study of injuries of the elbow joint, many mistakes were made in diagnosis, through lack of knowledge of the normal appearance of the joint at successive ages. Injuries about the elbow in children naturally present difficulties in diagnosis owing to the complexity of the structures involved. The bony structures contiguous to the

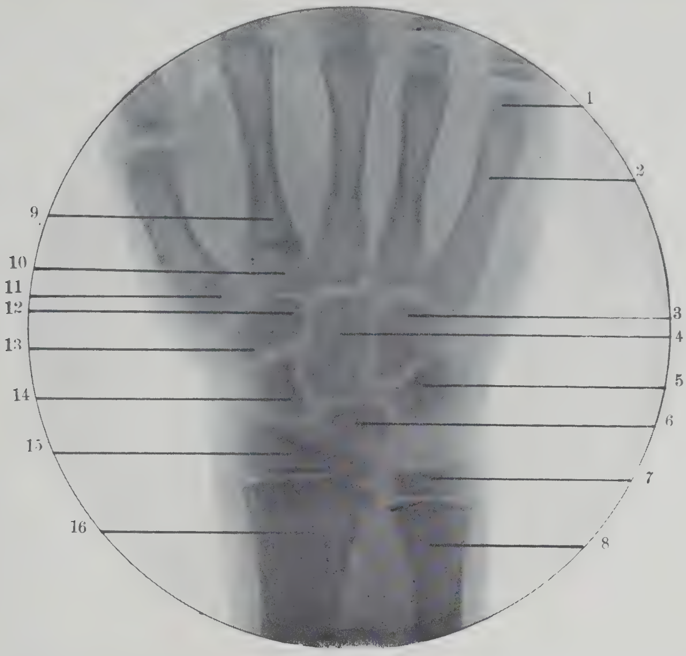


FIG. 150.—Wrist at Eleven Years. 1, Epiphysis of fifth metacarpal; 2, shaft of fifth metacarpal; 3, unciform; 4, os magnum; 5, cuneiform; 6, semilunar; 7, lower epiphysis of ulna; 8, ulna; 9, second metacarpal; 10, epiphysis of same; 11, epiphysis of first metacarpal; 12, trapezoid; 13, trapezium; 14, scaphoid; 15, lower epiphysis of radius; 16, radius. (Original.)

FIG. 151.—Wrist at Twelve Years. 1, Proximal phalanx of thumb; 2, epiphysis of same; 3, first metacarpal; 4, notch marking nearly completed union of second metacarpal and its epiphysis; 5, epiphysis of first metacarpal; 6, trapezoid; 7, trapezium; 8, scaphoid; 9, lower epiphysis of radius; 10, radius; 11, proximal phalanx of little finger; 12, epiphysis of same; 13, epiphysis of fifth metacarpal; 14, fifth metacarpal; 15, os magnum; 16, unciform; 17, cuneiform; 18, semilunar; 19, lower epiphysis of ulna; 20, ulna. (Original.)

joint develop usually from nine centres, which, however, are not always radiographically distinct. Cases have been recorded in which a fracture was supposed to have been present in an injured elbow, and the attending physician was misled by the radiograph, thinking that the epiphyseal lines were solutions of

Fig. 152.—Hand at Thirteen Years. 1, Epiphysis of fourth metacarpal; 2, fourth metacarpal; 3, unciform; 4, os magnum; 5, cuneiform; 6, pisiform; 7, lower epiphysis of radius; 8, lower epiphysis of ulna; 9, ulna; 10, radius; 11, shaft of first metacarpal; 12, epiphysis of same; 13, trapezium with coalescing shadow of trapezoid; 14, scaphoid; 15, semilunar. (Original.)

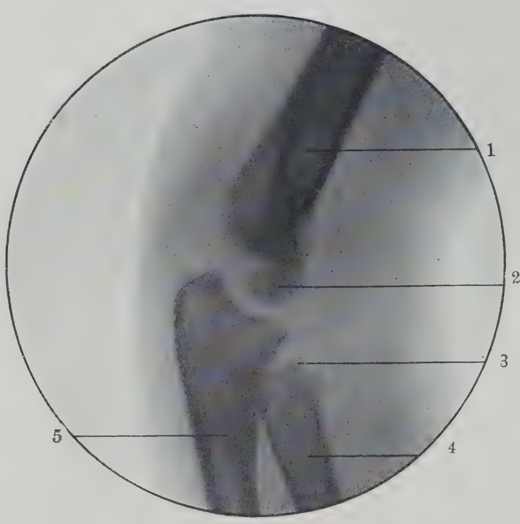


Fig. 153.—Radiograph Showing Lateral View of Elbow at Six Years. 1, Humerus; 2, capitellum; 3, upper epiphysis of radius; 4, radius; 5, ulna. (Original.)

continuity caused by violence. In the interpretation of radiographs of the elbow, greater care is necessary to secure proper reading of the appearances presented than in any other part of the body.

The first manifestation of an epiphyseal nucleus in the elbow is that of the

capitellum, which appears in the form of a little rounded knob usually during the second or third year of life.

In Fig. 153, which is a lateral view of the elbow at six years of age, the capitellum has attained considerable size, so that it projects somewhat into the

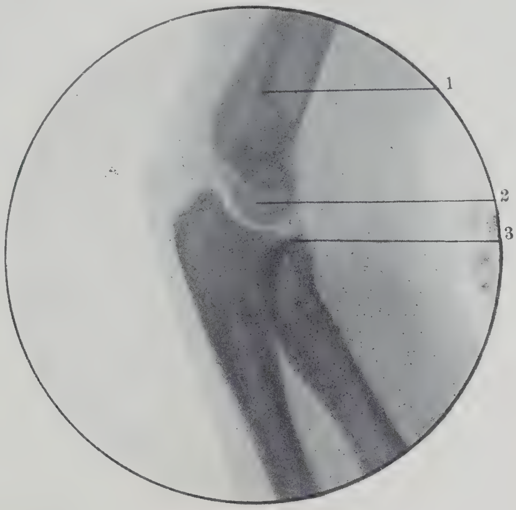


FIG. 154.—Radiograph Showing Lateral View of Elbow at Seven Years. 1, Humerus; 2, capitellum; 3, upper epiphysis of radius. (Original.)

rounded space formed by the greater sigmoid cavity. In this figure we note the appearance, as yet barely distinguishable, of the upper epiphysis of the radius, which shows itself first as a little button-like body.

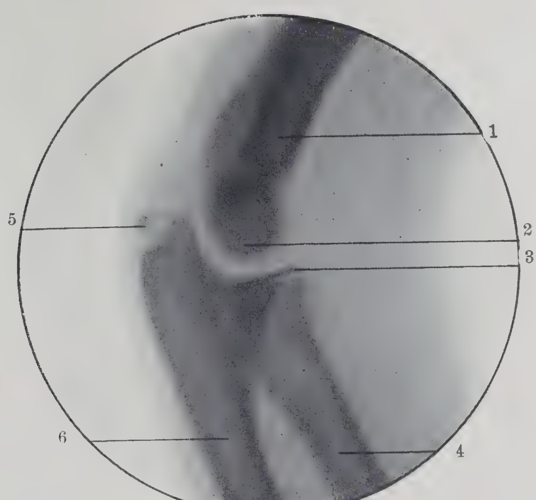


FIG. 155.—Radiograph Showing Lateral View of Elbow at Ten Years. 1, Humerus; 2, capitellum; 3, upper epiphysis of radius; 4, radius; 5, epiphysis of olecranon; 6, ulna. (Original.)

In Fig. 154 the capitellum occupies more of the sigmoid cavity while the upper epiphysis of the radius is much more distinctly visible.

In Fig. 155 we see at ten years of age the primary centre for the olecranon. The capitellum at this age has, superimposed upon it, the shadow of the trochlea, which, however, in lateral views of the joint, cannot usually be made out. The upper epiphysis of the radius has increased in size so that its diameter is about equal to the diameter of the shaft of the radius.

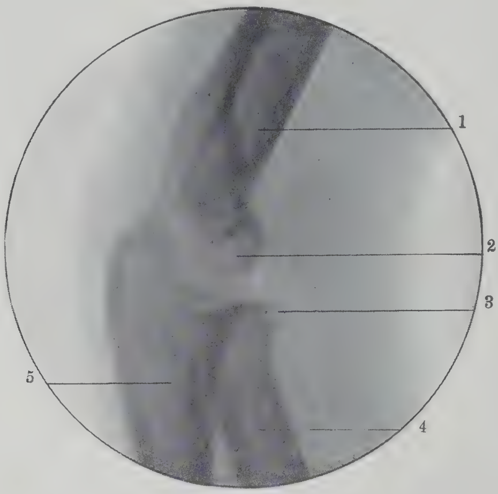


Fig. 156.—Radiograph Showing Lateral View of Elbow at Eleven Years. (Variation.) 1, Humerus; 2, capitellum; 3, upper epiphysis of radius; 4, radius; 5, ulna. (Original.)

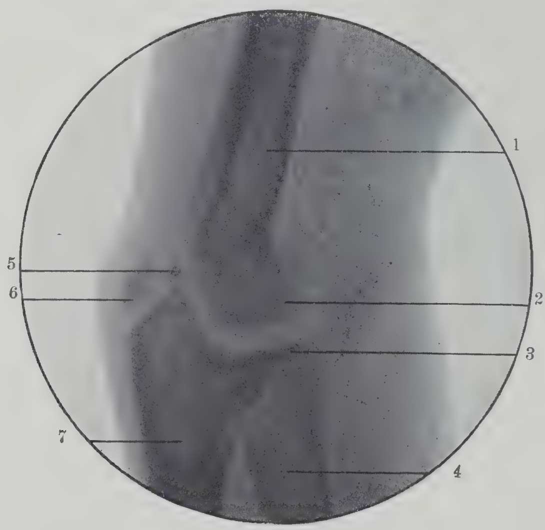


Fig. 157.—Lateral View of Elbow at Fourteen Years. 1, Humerus; 2, capitellum; 3, upper epiphysis of radius; 4, radius; 5, secondary centre for olecranon; 6, primary centre for olecranon; 7, ulna. (Original.)

Fig. 156 shows the elbow joint at eleven years of age. In this plate no trace of the olecranon is yet discernible. This must be considered simply as an irregularity of development in a particular individual. The other epiphyses show the degree of development normal for that age.

In Fig. 157, which is a lateral view of the elbow at fourteen, we find that the primary centre for the olecranon has greatly increased in size and that a secondary centre has appeared. These usually coalesce during the next six months, giving rise oftentimes to a peculiar elongated body. Here also the shadow of the trochlea is superimposed upon the shadow of the capitellum. The shadow of the trochlea is posterior to the shadow of the capitellum, while the shadow of the internal condyle cannot usually be differentiated in a lateral view. Their superimposed lines often make their recognition a matter of some difficulty.

In Fig. 158 we have a lateral view of the elbow at age fifteen, which shows the partial union of the olecranon to its shaft. The serrated line which marks the partial epiphyseal separation has been oftentimes mistaken for a fracture line. In two cases of injury which came under the writer's observation, the patients were compelled to wear splints for several weeks owing to the misinterpreta-

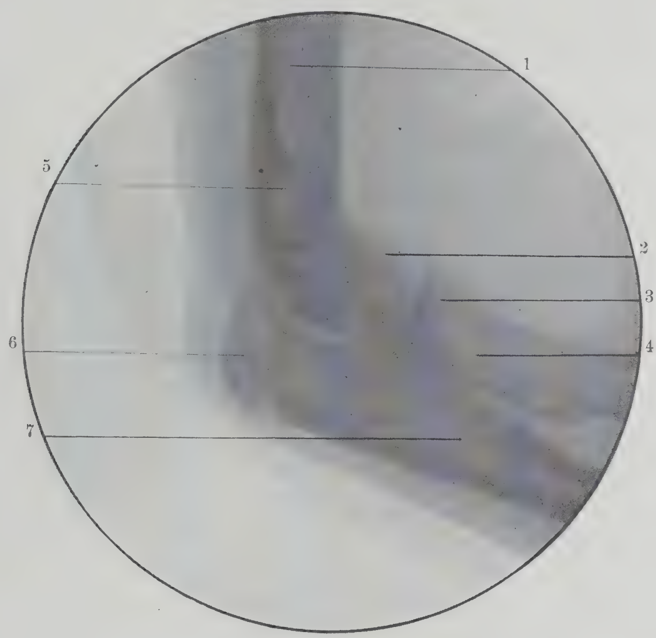


FIG. 158.—Radiograph Showing Lateral View of Elbow at Fifteen Years. 1, Humerus; 2, capitellum; 3, upper epiphysis of radius; 4, radius; 5, compact tissue of the lower end of humerus; 6, epiphysis of olecranon partial joint; 7, ulna. (Original.)

tion in this respect of the radiogram of the injured joint. At age sixteen the separation between the epiphysis belonging to the radius and its diaphysis is so slight as often to escape notice.

The vertical view of the developing elbow is perhaps more deceptive than the lateral views and requires study for its comprehension. In Fig. 159 is seen the elbow joint of a six-year-old boy. The capitellum is well formed while the small disc of the epiphysis of the radius is sharply shown.

In Fig. 160, which is a vertical view of the elbow at age ten, the centre for the

internal condyle is well formed. The capitellum is well shown; the superimposition of the trochlea over the shadow of the sigmoid cavity prevents its recognition.

In Fig. 161, which is a vertical view of the elbow at age eleven, we find the upper epiphysis of the radius remarkably well defined. The capitellum shows a

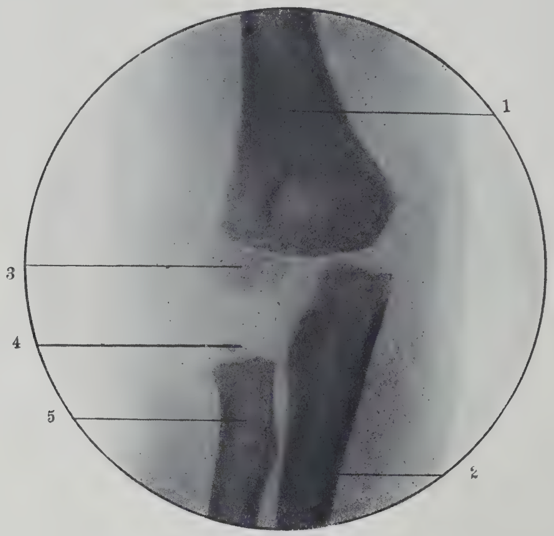


Fig. 159.—Vertical View of Elbow at Six Years. 1, Humerus; 2, ulna; 3, capitellum; 4, upper epiphysis of radius; 5, radius. (Original.)

Fig. 160.—Vertical View of Elbow at Ten Years. 1, Humerus; 2, trochlea; 3, capitellum; 4, epiphysis of radius; 5, radius (Original.)

partial attachment to the shaft, although the internal edge is distinctly separated. The olecranon fossa manifests itself as a rounded space of less density than the adjacent thicker and more compact bone tissue. At this age the centre for the internal condyle is also quite distinct from the diaphysis.

In Fig. 162—a vertical view of the elbow at age twelve—the internal condyle is partly attached to its shaft. Oftentimes in carefully prepared plates the epiphysis of the olecranon can be made out in the light shading of the olecranon fossa. On the external surface the capitellum has increased in size, and the

Fig. 161.—Radiograph Showing Vertical View of Elbow at Eleven Years. 1, Humerus; 2, olecranon fossa; 3, capitellum; 4, upper epiphysis of radius; 5, radius; 6, trochlea; 7, ulna. (Original.)

Fig. 162.—Radiograph Showing Vertical View of Elbow at Twelve Years. 1, Capitellum; 2, upper epiphysis of radius; 3, radius; 4, humerus; 5, trochlea; 6, ulna. (Original.)

trochlea can often be made out with its shadow superimposed on that of the sigmoid cavity. The upper epiphysis of the radius is still ununited.

In Fig. 163, which is a vertical view of the elbow at age thirteen, we find the centre for the olecranon manifesting itself more distinctly through the shadow of the olecranon fossa. The peculiar appearance of the capitellum should be

Fig. 163.—Radiograph Showing Vertical View of Elbow at Thirteen Years. **1, Trochlea; 2, ulna;** 3, humerus; 4, capitellum; 5, upper epiphysis of radius; 6, radius. (Original.)

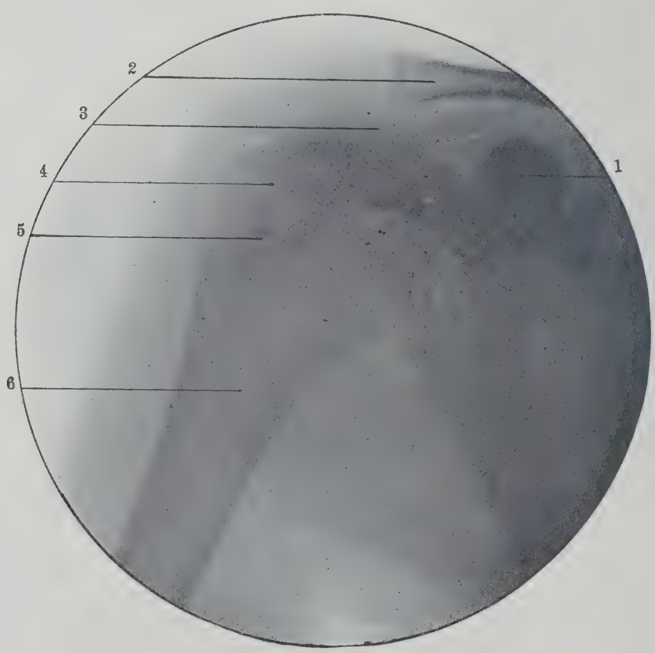


Fig. 164.—Shoulder Joint at Eleven Years. 1, Coracoid process; 2, distal extremity of clavicle; 3, acromion process; 4, head of humerus; 5, epiphyseal line; 6, shaft of humerus. (Original.)

noticed, since on its upper and outer border we find an irregular projection which marks the extension of the bony structure in its endeavor to bridge across the intervening space. On the internal side the internal condyle is partly

FIG. 165.—Radiograph Showing Hip Joint at Seven Years. 1, Ischium; 2, head of femur; 3, centre of greater trochanter; 4, femur. (Original.)

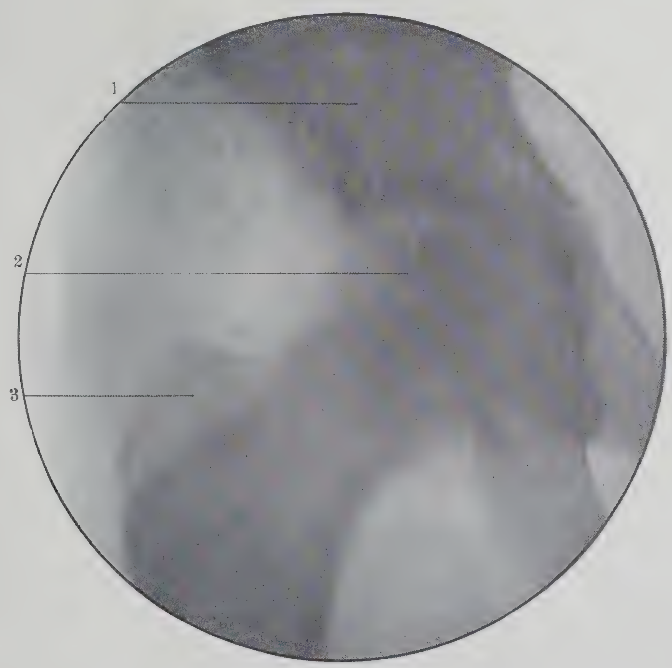


FIG. 166.—Hip Joint at Eleven Years. 1, Ilium; 2, head of femur; 3, great trochanter. (Original.)

attached to its shaft. At this age the trochlea can usually be made out quite distinctly.

The radiographic appearance of the shoulder joint during the development of the child does not usually vary, and presents no particular difficulties of interpretation. In Fig. 164, which was made from an eleven-year-old boy, the epiphyseal line separating the head of the humerus from the shaft is distinctly seen. This ordinarily persists until about the nineteenth or twentieth year.

In Fig. 165, which represents the hip joint of a child of seven, we find the head of the femur quite distinct. In the interpretation of radiographs of congenital dislocation of the hip it is important to remember that the first bony appearance of the head of the femur ordinarily occurs at the end of the first year of life. In the same figure we see the separate centre for the great trochanter, which makes

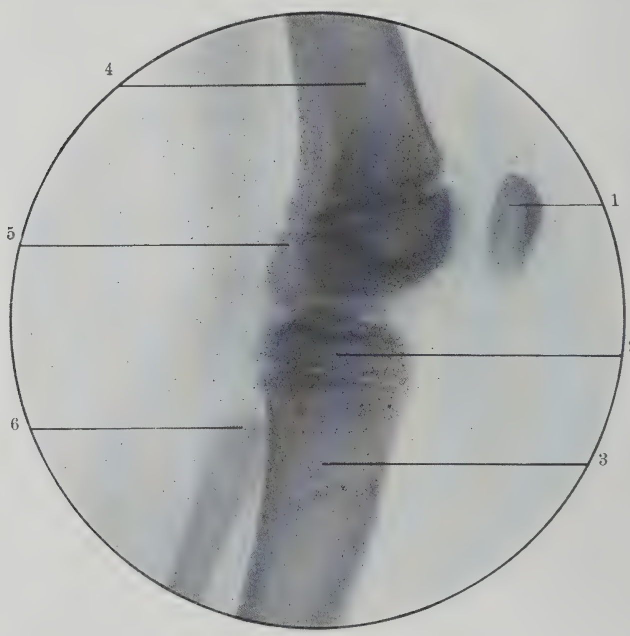


Fig. 167.—Radiograph Showing Lateral View of Knee Joint at Seven Years. 1, Patella; 2, upper epiphysis of tibia; 3, tibia; 4, femur; 5, lower epiphysis of femur; 6, upper epiphysis of fibula. (Original.)

its appearance at widely varying periods, sometimes as early as the fourth year, and again, in some cases, not until the eighth year. The lesser trochanter generally makes its first appearance at a much later date, usually about the eleventh or twelfth year.

In Fig. 166 the greater trochanter (at age eleven) is most distinctly shown. It usually unites with the shaft at about the same time as when the epiphyseal line between the head of the femur and the shaft disappears; namely, about the eighteenth year.

The lower epiphysis of the femur is shown in Fig. 167, which is a lateral view

of the knee joint at seven years of age. The patella is a bone which radiographically appears at different ages—usually after the third or fourth year; it presents the form of a rounded shadow without the well-defined angles which later distinguish it.

The upper epiphysis of the tibia, as well as the upper epiphysis of the fibula, usually appears during the second year of life. The development of these epiphyses and the change in shape of the patella are illustrated in Fig. 168, which is a lateral view of the knee at age eleven. During the next year the upper epiphysis of the tibia throws out, from its lower part, a projection which is fashioned

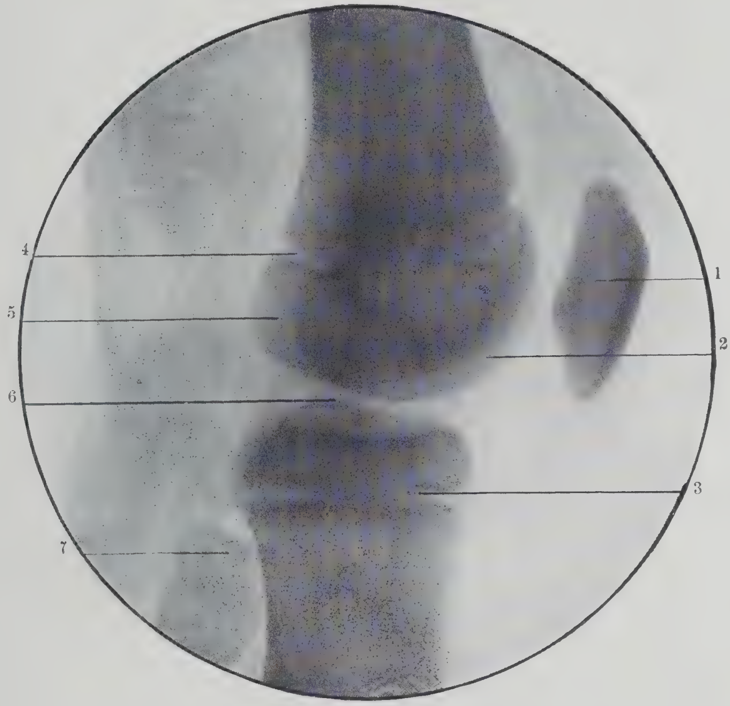


FIG. 168.—Lateral View of Knee Joint at Eleven Years. 1, Patella; 2, inner condyle; 3, epiphyseal line at upper end of tibia; 4, epiphyseal line between the femur and the outer condyle; 5, outer condyle; 6, spine of tibia; 7, upper epiphysis of fibula. (Original.)

somewhat like a tongue—a projection which afterward becomes the tubercle of the tibia.

The antero-posterior view of the knee joint is shown in Fig. 169, which was made with the posterior surface of the joint next to the photographic plate. The distance of the patella from the plate increases its apparent size, so that its shadow is less distinct and can scarcely be differentiated from the lower end of the tibia. The spinous process of the tibia is shown as it projects into the joint space. The epiphyseal lines are so distinct and regular that their appearance is not likely to lead to a misinterpretation.

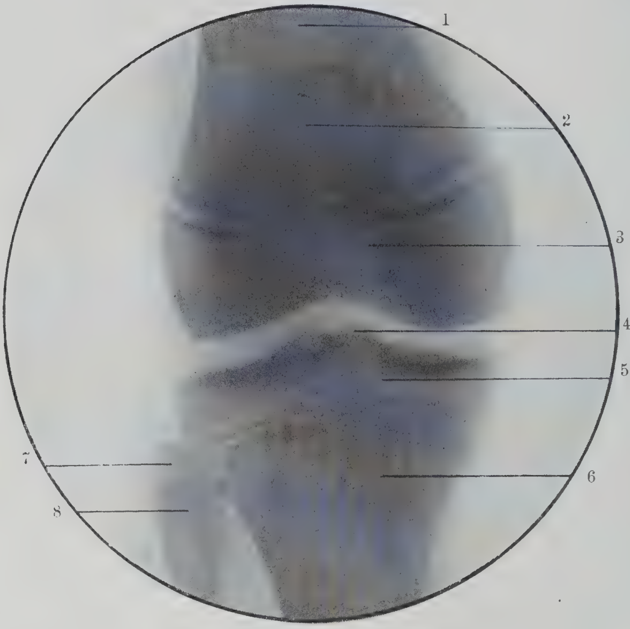


FIG. 169.—Radiograph Showing Vertical View of Knee at Eleven Years. 1, Femur; 2, patella; 3, lower epiphysis of femur; 4, spine of tibia; 5, upper epiphysis of tibia; 6, tibia; 7, upper epiphysis of fibula; 8, fibula. (Original.)

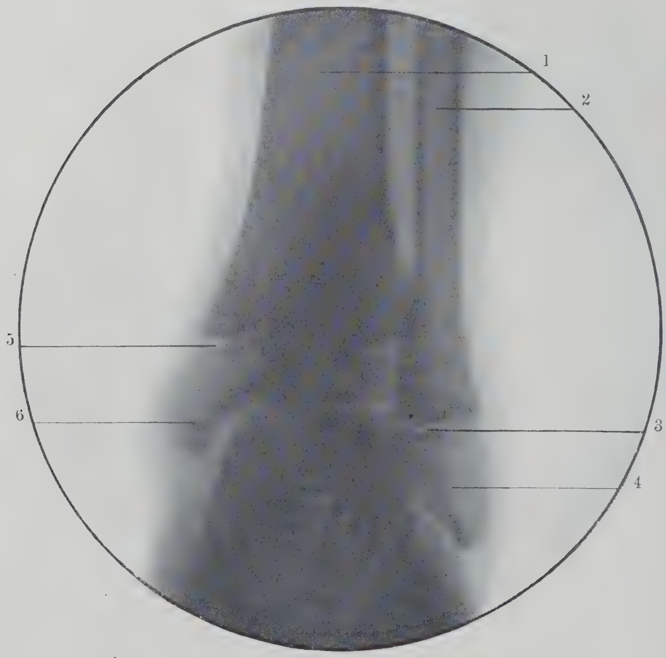


FIG. 170.—Vertical View of Ankle Joint at Eleven Years. 1, Tibia; 2, fibula; 3, epiphyseal line of fibula; 4, lower epiphysis of fibula; 5, epiphyseal line of tibia; 6, lower epiphysis of tibia. (Original.)

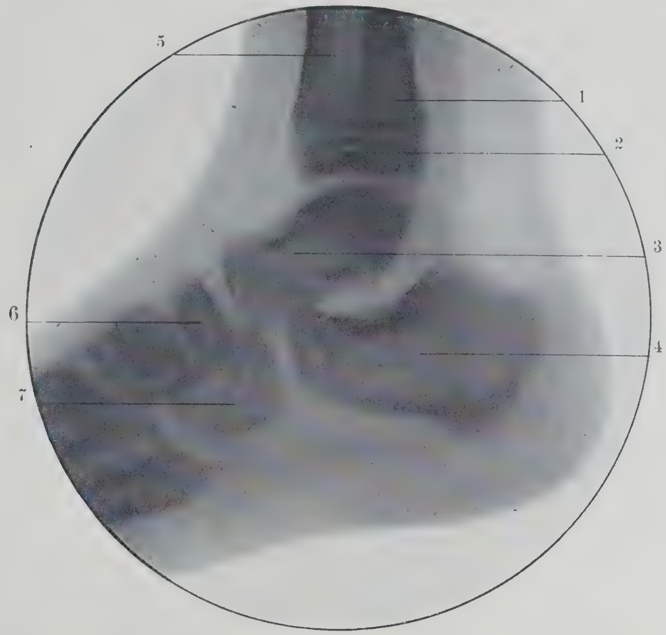


Fig. 171.—Radiograph Showing Lateral View of Ankle at Seven Years. 1, Superimposed shadow of fibula; 2, lower epiphysis of tibia; 3, astragalus; 4, os calcis; 5, tibia; 6, scaphoid; 7, cuboid. (Original.)

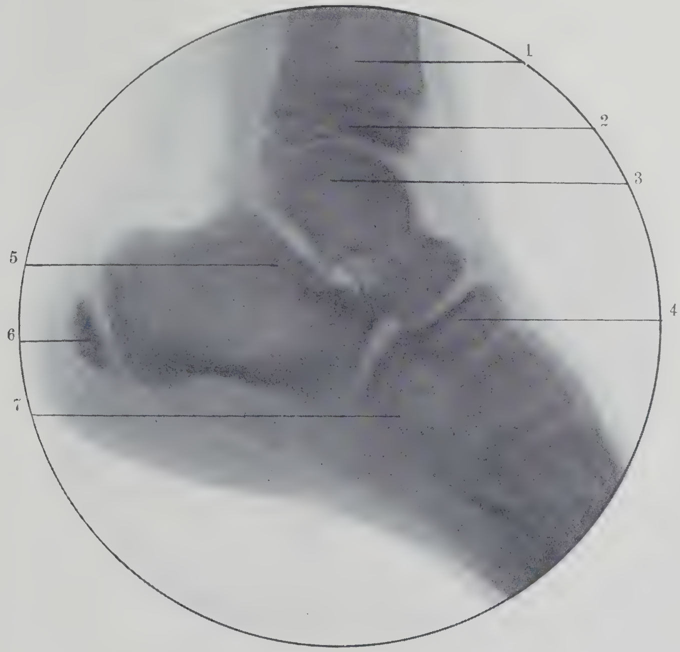


Fig. 172.—Lateral View of Ankle at Eleven Years. 1, Tibia; 2, lower epiphysis of tibia; 3, astragalus; 4, scaphoid; 5, os calcis; 6, epiphysis of os calcis; 7, cuboid. (Original.)

Fig. 170 is a vertical view of the ankle at age eleven; the lower epiphysis of the fibula, as it forms the external malleolus, is a well-known object. On the internal side the broad epiphysis of the tibia is shown above the talo-crural joint.

In Fig. 171, which is a lateral view of the ankle joint at seven years of age, the distance between the tarsal bones is strikingly obvious. At birth the tarsus usually shows a small centre for the os calcis and one for the astragalus. The cuboid appears during the fifth month, while the scaphoid is first seen about the fourth year. The considerable space which is present between the bony

Fig. 173.—**Radiograph Showing Lateral View of Ankle at Twelve Years. 1, Scaphoid; 2, internal cuneiform; 3, cuboid; 4, tibia; 5, lower epiphysis of tibia; 6, fibula; 7, astragalus; 8, os calcis; 9, epiphysis of os calcis.** (Original.)

centres of the tarsal bones before the age of five explains at a glance why moulding operations upon the foot at this age are possible.

The epiphysis of the os calcis appears ordinarily at the eighth or ninth year. In Fig. 172, which is a lateral view of the ankle at age eleven, this epiphysis is shown in an already well-advanced state of development, with fine osseous deposits between its main body and the os calcis.

Further development of this epiphysis occurs during the following year, as is shown in Fig. 173.

In Fig. 174, which gives a lateral view of the ankle at age thirteen, we have the partial union of the epiphysis with the os calcis. This union may take place a

FIG. 174.—Radiograph Showing Lateral View of Ankle at Thirteen Years. 1, Tibia; 2, fibula; 3, lower epiphysis of tibia; 4, astragalus; 5, scaphoid; 6, internal cuneiform; 7, cuboid; 8, lower epiphysis of fibula; 9, os calcis; 10, epiphysis of os calcis. (Original.)

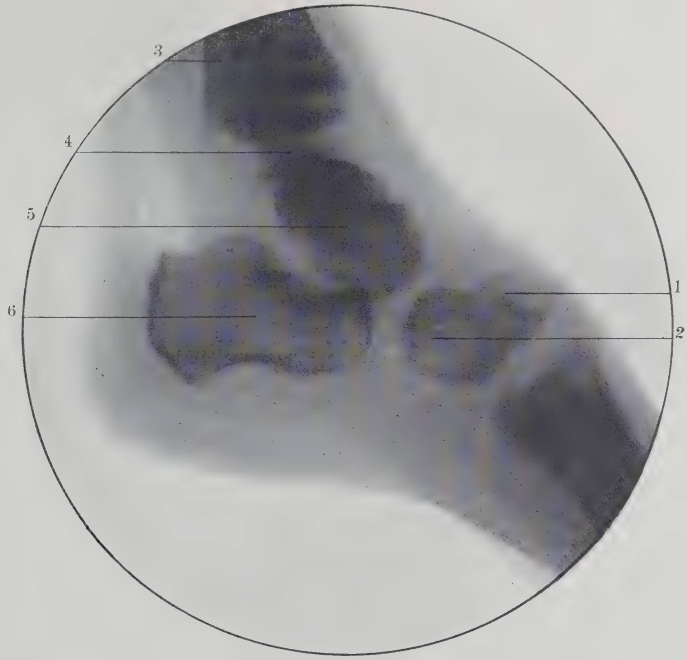


FIG. 175.—Radiograph Showing Lateral View of a Cretin's Foot at Twelve Years. 1, Scaphoid: 2, cuboid; 3, tibia; 4, fibula; 5, astragalus; 6, os calcis. (Original.)

year or so later. In this radiogram the distance between the tarsal bones approaches that of the adult foot.

The general development of the child may oftentimes be inferred with a fair degree of accuracy from the degree of development of the osseous framework. In Fig. 175, which is a lateral view of the ankle of a cretin—twelve years of age,—we find the bones showing the degree of development which we should ordinarily find at four years. The mental development of the patient corresponded to the intelligence which we should expect to find in a child of five years.

In interpreting radiographs of injured joints of children, as the writer has insisted above, some experience is necessary. We should be familiar with the appearances ordinarily presented at different ages, and we should be able promptly and correctly to interpret the exceptions which may occur. Ordinarily, it is a safe rule, in cases of obscure injury, particularly about the elbow joint, to make a second radiograph, at the same time, of the uninjured joint, for the purpose of comparing it with the radiograph of the injured joint, both of the pictures to be taken under the same conditions.

It has been the experience of radiographers that epiphyseal separations are rarely met with. The clinical diagnosis of a traumatic separation of the epiphysis will often be found to be inaccurate, as the radiograph usually shows a solution of continuity through the bony structures adjacent to the epiphyseal line. The sudden violence at the time of injury seems to snap the bone in preference to pulling apart the more yielding soft tissues. This is perhaps fortunate, as the readjustment of the bony fragment can be better effected than the replacement of the unossified structures nearer the joint.

THE TECHNIQUE OF RADIOGRAPHIC WORK AS APPLIED TO SURGERY, AND THE INTERPRETATION OF RADIOGRAPHS.

By Mr. WALTER J. DODD and ROBERT B. OSGOOD, M.D., Boston, Mass.

I. RADIOGRAPHIC TECHNIQUE

The development of x-ray work since Roentgen's discovery has been rapid and the broadening of its field of usefulness has been great. A short decade has seen the interesting plaything of surgery absorb the attention of earnest scientific workers until many men have sacrificed the general practice of medicine to become specialists in radiology, or even to devote themselves exclusively to certain branches of the art.

It is not the purpose of this article to consider in great detail the technique and apparatus necessary for these special x-ray investigations, but rather to outline with sufficient clearness apparatus and methods which, if obtained and followed, will allow a busy practitioner to install and operate an x-ray plant with satisfaction and accuracy.

In large centres it is now possible to obtain x-ray work which is up to the best modern standards. Its field of usefulness, however, is by no means confined to these large centres, and in many instances the advantages of office plants and personal operation far outweigh the value of these larger x-ray laboratories.

We wish it distinctly understood that the apparatus here suggested and the methods advised are simply those which, from practical experience, we know to be accurate and with which work of high quality can be done. Where several forms of apparatus, of nearly equal value, have been devised, the simplest have invariably been chosen for description. The methods advised should be considered for the most part as working bases from which the practitioner beginning x-ray work may start, later elaborating and adapting them to his individual needs.

To the interpretation of x-ray plates far less space has been given than the importance of the subject justifies. The writers believe that here again the aim of the work should be to suggest broad diagnostic points, feeling sure that the power of finer discrimination can come satisfactorily only through large experience and patient comparison with the normal.

In the description of the characteristics of the various conditions which will

be considered later, we presuppose good and even lighting of the plates, such as is shown in Fig. 176. This illuminator will be subsequently described.

We wish to acknowledge our indebtedness to a valuable and exhaustive unpublished paper by Dr. E. A. Codman, of Boston, and to the work of Schuchardt, "Die Krankheiten der Knochen und Gelenke" ("Deut. Chir.," Bd. xxviii.).

We shall consider the evidence of the various diseases in the skiagraph alone without discussing the histologic and pathologic changes or the detailed etiologic factors.

The therapeutic uses of the *x*-ray have been exhaustively treated in available books. We do not feel qualified to speak authoritatively on the technique of these measures, nor of their value. Pure surgery concerns itself little with most of the diseases commonly so treated. While recognizing differences of opinion among careful observers, we personally feel that *x*-ray therapy, except

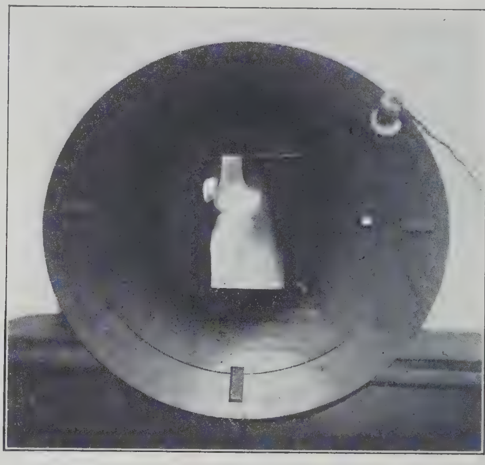


Fig. 176.—Illuminator. Light, evenly diffused, reflected from white back of box.

in the superficial forms of epithelioma, should never take the place of surgery in operable cases of cancer. Although differences of opinion also exist as to the value of *x*-ray treatment following operations for carcinoma, the evidence in our minds seems to warrant its use as a post-operative safeguard against recurrence. In the keratoses and superficial epitheliomata the results of *x*-ray treatment are certainly comparable with the surgical procedures. In the treatment of lupus and the superficial forms of tuberculosis, it undoubtedly offers us the best method at our disposal and shows results little short of marvellous in the light of our old conceptions.

Measurers of the intensity and the quality of the rays are to be obtained in varied forms of greater or less accuracy. Some of these meters should be used in therapeutic work, and the treatment standardized as much as possible.

A knowledge of electricity can never be other than a help in radiology. We do not deem, however, an intimate acquaintance with the subject essential to

practical x-ray work. The basic principles must be known; to be an electrical engineer is unnecessary. It will be taken for granted that the principles of conduction and induction are in a general way understood, and the component parts of plants adapted to different forms of current, or to an entire absence of available current, will be described. The following list comprises what we consider the essentials of an x-ray plant:

Coil;

Interrupter;

Several good x-ray tubes;

Rheostat;

Switchboard with ammeter for primary circuit, fuse of lower reading than main fuse, and knife switches;

Compression cylinder;

Adjustable table or support for patients;

Illuminating lantern;

Solid and well-constructed tube holder.

Coil.

The plant about to be described has been submitted to the severe test of hospital as well as large office practice. No originality of method or apparatus is claimed, the purpose of the writers being to describe apparatus and technique that have proved to be practical, simple, and satisfactory.

In order to do quick radiographic work the coil should give a discharge of at least 4 to 8 inches. It is not simply length of spark that is needed, but

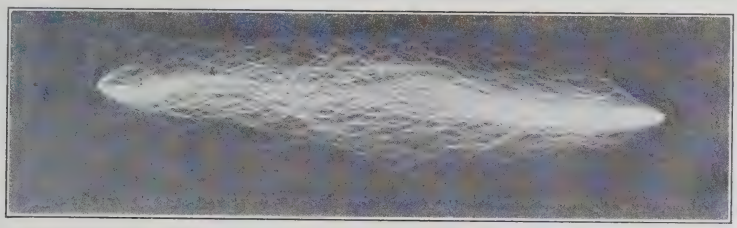


Fig. 177.—Illustrates spark spoken of as *thin spark*. Impossible to get higher reading than 3 milliamperes with this coil; from 1½ to 2 milliamperes being the average reading with 16 amperes on primary circuit. Length of spark 8 inches. Exposure for hip, in the case of an adult weighing 160 lbs., from 2 to 3 minutes. (Original.)

volume. A coil that will give a long, thin 12-inch spark is not to be compared with one that will give a flaming 8-inch spark. The flaming discharge indicates quantity, and quantity is essential to speed, and speed is essential to good work. By speed we do not mean snap-shots. We must keep in mind the fact that we are not dealing with light from one source, but that the x-ray light gives rise to other radiations, all of which have more or less influence on a pho-

tographic plate. For this reason we must strive to get, in as short a time as is consistent with good work, the greatest quantity of x-ray light, thereby eliminating secondary radiations as much as possible The coils used by the writers are capable of giving a 6- to 10-inch flaming discharge (Figs. 177 and 178) when used with a Wehnelt interrupter; the ammeter in the primary reading 10 amperes, and service being derived from a direct current of either 220 or 110 volts. The primaries of these coils are so wound that self-induction has been greatly increased over that of the primaries used when the electrolytic interrupter was first introduced. It is to Dr. Walters, of Hamburg, that the credit of variable primary

Fig. 178.—Illustrates flaming discharge, 8 inches in length. Meter will read 10 milliamperes with 10 amperes on primary circuit. Exposure for hip, in an adult weighing 160 lbs., 20 to 40 seconds. (Original.)

inductance should be given. Such winding is absolutely essential to good results with the Wehnelt interrupter.

All large coils of first-class make are now equipped with primaries of this type and are most highly recommended. Coils of first-class manufacture will always give the spark at which they are rated, but the operator is advised not to submit the coil to such strain. Never spark the coil to its full capacity. You can test a 12-inch coil without forcing the discharge through the air gap of 12 inches. To test a coil of 12-inch rating the writers place the discharge rods about 8 inches apart and slowly turn on the current. If the coil is in good working order, sparking will begin with a thin, continuous, snappy spark which is thicker and broader at one end. This is the cathode or negative terminal of the coil and should be marked with the negative sign. As more current is allowed to flow in, the discharge increases until finally we get the yellow con-

tinuous spark. Never test the coil by sparking unless it is absolutely necessary, as the coil is likely to become overheated and affect the insulation of the secondary circuit.

Interrupter.

The purpose of the interrupter is to break the primary current rapidly and completely, while at the same time the period of make is long enough to saturate the primary coil. Mechanical interrupters of modern pattern have reached a high stage of perfection and are heartily endorsed by many, but the current-carrying capacity of all mechanical interrupters is low and the time of exposure must be long when compared with an interrupter of high-current-carrying capacity, such as the liquid electrolytic, mercury, and Wehnelt. As the writers believe from their own experience that the Wehnelt is by all means the most satisfactory for good radiographic work that type alone will be described.

The action of the electrolytic interrupter is brought about by electrolysis of acidulated water, gas forming at the anode or positive plate, which gas for an instant envelops that plate and momentarily breaks the current; a very high rate of interruption may thus be obtained. The electrolytic or Wehnelt interrupter consists of a lead plate or tube, which must always be connected to the negative terminal of the main current, and a platinum point which must always be made positive. These are then placed in a vessel of glass or earthenware containing sulphuric acid, specific gravity 1.20; the platinum point is usually passed through a glass or porcelain tube, the strength of current being dependent upon the amount of platinum surface projecting through the tube into the liquid. As the amount of current depends on the amount of platinum surface thus exposed, we can readily see that we have at hand an interrupter the current-carrying capacity of which is almost unlimited and far beyond what it is possible to use with our present knowledge of tubes and coils. The Wehnelt interrupter was made practicable for radiographic work by Dr. Walters' improvement in induction-coil construction whereby the self-induction of the primary could be raised by using two or more layers of wire according to the vacuum and spark resistance of the tube. It is now possible to use the Wehnelt interrupter when employing tubes for therapeutic purposes. By raising the self-induction we are enabled to use tubes of much lower vacuum; and the writers have used such tubes for over twenty minutes' continuous operation with three amperes on the primary circuit, the spark length of the coil being a heavy 10-inch flaming discharge when used with one layer of primary, whereas with two layers of primary on this particular coil it is impossible to get a flaming discharge of more than three and one-half inches in length even with fifteen amperes on the primary circuit. It is interesting to note that the character of the spark is almost completely changed when the self-induction is raised. It is much shorter, but at the same time much thicker (Fig. 177). This

fact may be taken advantage of to operate tubes of very low resistance, and at the same time get good radiographic results, although the exposure is invariably longer. This discovery, made by Dr. Walters, is one of the greatest steps in advance, it being now possible to take advantage of that most efficient and admirable interrupter, the Wehnelt electrolytic. It is now possible by means of this interrupter and coils of modern construction to get good radiographs of the deeper parts of the body in a few seconds—a very brief time when compared with the period of several minutes required by the older apparatus.

The interrupter used by the writers consists of a piece of lead pipe one and three-fourths inches inside diameter, three-sixteenths inch thick, and about twelve inches long. This is fastened to a board by cutting two ears or lugs, bending them back and screwing the same to the board. A hole is now cut

Fig. 179.—Interrupter Board with Lead and Glass Tube attached. The spiral wire, 1, is attached to the lead pipe. The loop, 2, is attached to the platinum wire. A slot is cut in the lead pipe close to the board for the outlet of the glass tube, which is inside the lead pipe. The latter is 12 inches long, with an inside diameter of $1\frac{1}{2}$ inches. Liquid in stone crock comes to within one inch of outlet of glass tube. (Original.)

through the board corresponding to the centre of this lead tube (Fig. 179). Into this hole a glass tube ten inches long and one-half inch inside diameter is placed. To the lower end of this tube a capillary tube one and one-half inch long is sealed, this capillary being for the platinum wire to pass through. This tube has a side outlet, two inches from the top, to allow the liquid, which always rises in the tube when the interrupter is running, to pass out, otherwise it would overflow on to the cover. A copper wire, sixteen inches long, is now taken, and to one end of it a piece of iridized platinum four inches long is fastened. By flattening the end of the copper wire slightly, a small hole may be bored in it and the platinum wire passed through, bent over, and then soldered securely in place. The copper wire is passed through a cork, which is fitted tightly into

the glass tube. Special care should be taken to prevent the platinum from coming in contact with the lead pipe.

The board with the tube attached is now put in a stone crock large enough to hold from one to six gallons. Six-gallon stone crocks are used with the interrupter illustrated in Figs. 179 and 180; and the fluid to be used is either sulphuric acid, specific gravity 1.20, or a half-saturated solution of magnesium sulphate acidified with sulphuric acid. This magnesium-sulphate solution was suggested by E. Hauser, of Madrid, and has been used at the Massachusetts General Hospital for eight months without being renewed. The objection to the Wehnelt interrupter, on the ground that the solution gets overheated (that is, above 90° F.), seems to be obviated to a great extent by means of this solution; for we have employed it day after day, and on testing have found it to be 115° F., which high temperature does not interfere with its efficient action.

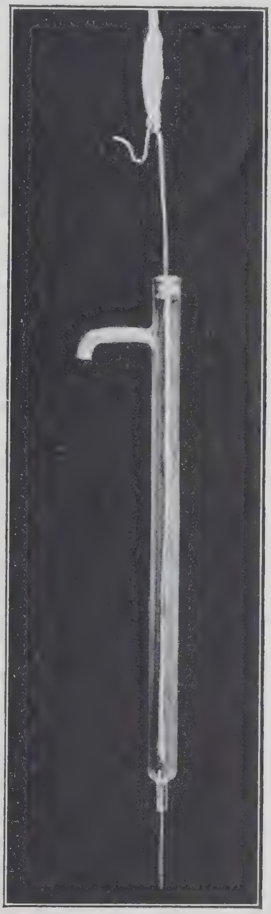


FIG. 180.—Interrupter tube. Length, $10\frac{1}{2}$ inches; inside diameter, $\frac{1}{2}$ inch. To the larger tube is sealed a capillary tube, $1\frac{1}{2}$ inch long, through which a platinum wire slides. This capillary tube is large enough to carry the platinum wire without friction. (Original.)

The acid or the magnesium-sulphate solution used in the interrupter should be covered with some heavy oil, such as dynamo oil, to the extent of about one-eighth of an inch deep. This prevents evaporation of the liquid and also the spraying which always occurs to some extent when heavy currents are used.

As before stated, the amount of current passing into the primary circuit is dependent upon the amount of platinum or anodal surface that comes in contact, through the glass tube, with the liquid. On the coil used at the Massachusetts General Hospital, where a direct current of two hundred and twenty volts is used, one-quarter of an inch of platinum of the size one forty thousandth gives three and one-half amperes; three-quarters of an inch will give eleven amperes; and one inch fifteen amperes. These figures cannot be taken as a guide, however, because so much depends on the resistance in the primary as well as in the secondary circuit.

To connect up the Wehnelt interrupter it is necessary to determine the positive terminal of the mains. To do this, we advise taking a dilute solution of potassium iodide, moistening a piece of filter paper with it, then bringing both wires into contact with the filter paper, great care being taken not to allow the wires to touch each other. If a dilute solution is used the iodine will be liberated

and a blue color will become evident at the positive pole. If, however, a strong solution is used a brown color will appear at the positive pole and sometimes this may be confused with the charring of the paper which occurs quite frequently at the negative pole. For this reason a dilute solution is advised. After it has been determined which is the positive pole it should be connected directly to the platinum terminal of the interrupter. The other, or negative, terminal is connected to the primary of the coil, the other end of the primary being connected to the lead of the interrupter (see Fig. 179). The interrupter having now been connected at all points, it is necessary to find out the amount of amperage that will be needed for operating each

FIG. 181.—*X*-ray Room of Massachusetts General Hospital, showing closet containing interrupters; on top of closet is the switchboard with knife switches, ammeter, and fuses. Fuses are of lower resistance than main fuses, so that when fuse blows out it may be replaced without the annoyance of going to main board, which may be in the cellar or at some distance. The operator works behind a screen, which, in this picture, has been pushed to one side. (Original.)

particular apparatus. From experiments on several coils we have ascertained that from twelve to fifteen amperes, with two hundred and twenty or one hundred and ten volts of a direct current, will be found sufficient for all radiographic work, a rheostat being used to control the amount of current flowing into the primary circuit. A lower voltage, if available, is recommended, from sixty to ninety volts being considered the best.

The crude interrupter described above is by no means meant to take the place of the more elaborate and easily adjusted apparatus now on the market, but its simplicity of construction and the fact that we have had several inter-

rupters of this type in use for over a year justify us, we think, in furnishing dimensions (Figs. 179 and 180).

We have spoken of the rheostat controlling the amount of current that goes into the primary circuit, and we feel that we are justified in saying that without it we cannot get the best results obtainable from an efficient and always

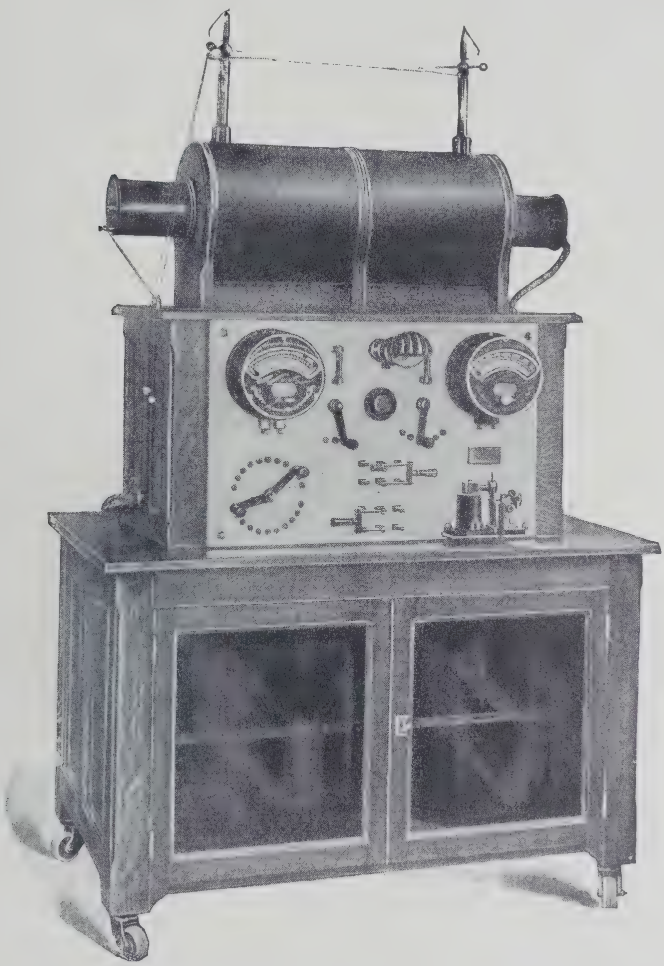


FIG. 182.—Coil showing mechanical interrupter, also switchboard with switches, rheostat, and meters. Wehnelt interrupter can be placed in closet below. The writer believes that a better arrangement is to have the switchboard at a distance from the coil and tube stand. By this plan the operator may always have the protective screen interposed between him and the active tube. (See also Fig. 181.)

satisfactory apparatus. To have sufficient platinum exposed in the liquid to give fifteen amperes in the primary circuit, without a rheostat to control it, is, to say the least, a somewhat dangerous proceeding; for, although the spark from a large induction coil may not cause harmful effects if received by the patient, it is certainly sufficient to make him hesitate as to the advisability of again submitting himself to such an examination. The rheostat gives the

operator complete control of the current passing into the coil. By this means he can use more or less according to the vacuum of the tube; moreover, if he uses the rheostat instead of the switch when shutting off the coil, all danger of the patient getting a shock is obviated. In our work we are accustomed to use a twenty-ampere rheostat with one-half or one-ampere steps. With this fine gradation the operator has great control over the amount of current going into the primary circuit. (Figs. 181 and 182.)

If the alternating current is the source of supply, a coil may be operated to good advantage by using the Wehnelt interrupter, or better by using in series with this interrupter the aluminum rectifier, thus converting the alternating into the direct current. It is not quite so easily managed as a coil operated on the direct current, but, if care be taken to keep the twenty-per-cent aqueous solution of ammonium phosphate alkaline, good results may be readily obtained from this rectifier. If expense is not to be considered, a motor converter is to be by all means recommended. Storage batteries may be used provided they be large enough to give sixty volts or over. For this purpose the storage battery of an automobile may be utilized, and the aluminum rectifier may be used for charging the storage battery. Many operators prefer the static machine, and there is certainly much to be said in its favor. In order to get good results from static machines a large one capable of being driven at a high rate of speed is necessary. This type of apparatus has been greatly developed since the advent of Roentgen's discovery, and the objection to the influence of atmospheric conditions has been in large measure eliminated. The static machine will give beautiful radiographs, is by no means as hard on the tubes, and is much simpler to operate. The objection to it is the liability, in damp weather, or when operated in certain climates, of failure to start even after much manipulation. Another objection is the time of exposure required. Much longer exposures are necessary than with the coil. Still, many operators prefer it to the coil and get beautiful results.

Tube.

The type of tube should be adapted to the machine. It is advisable to use small tubes on coils of short spark length, and large tubes on coils giving long, heavy discharge. The tubes used by the writers are of the Gundelach, Voltholm, and Friedlander type.

To accurately describe the appearance of an *x*-ray tube as the process of seasoning or ageing progresses seems almost impossible. The illustrations used by the writers (Plate V.) are intended to emphasize the more important visible changes that the tube undergoes during the time of ageing, which ageing we believe to be essential to good results.

When an *x*-ray tube is used for the first time it usually is of high resistance,

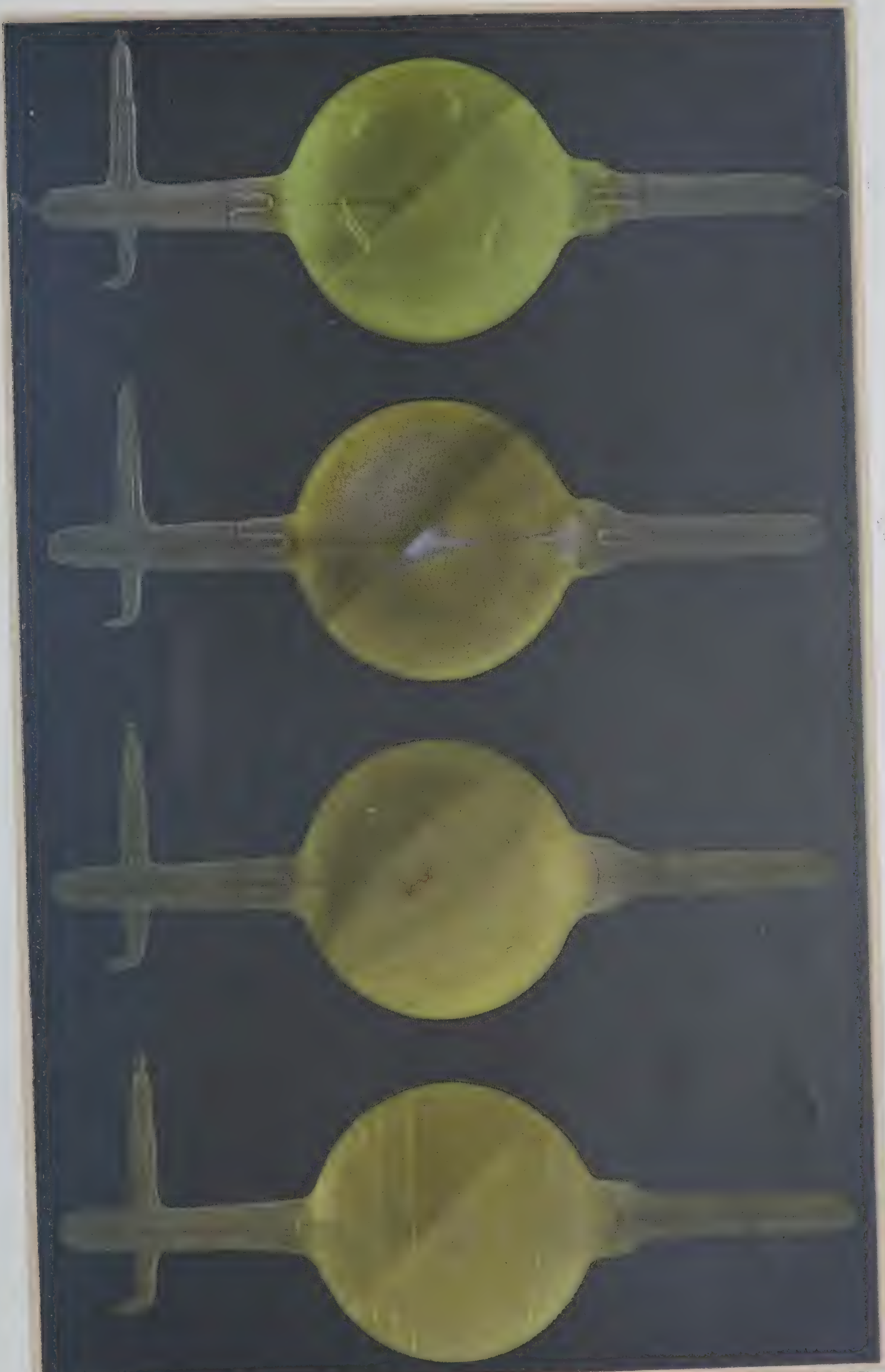
EXPLANATION OF PLATE V.

FIG. 1.—Illustrates an Unseasoned High-Vacuum Tube. With 12 amperes on primary circuit only 0.5 to 1 milliammeter can be forced through the tube. Radiographs taken under such conditions give thin plates showing practically no contrast between bone and flesh. Lowering the vacuum of this tube will increase the radiographic value, but only temporarily, as the tube is unseasoned, and will soon, even with short exposures, present an appearance such as is illustrated in Fig. 2. A seasoned tube may present an appearance similar to this.

FIG. 2.—Illustrates a Low-Vacuum Tube. The tube, under such conditions, is of very little value for radiographic work. It is *very important* to note that when the tube is in this state, *showing cathode stream,* we get a *very high reading* on milliammeter with comparatively *little energy* flowing into the primary circuit. This may be due not so much to the degree of vacuum as to what gases the vacuum is composed of. In unseasoned tubes we have gases driven off from terminals, whereas in seasoned tubes the vacuum is reduced by means of the regulator.

FIG. 3.—Illustrates the Appearance of the Tube when Working to the Best Advantage. Notice the illuminated spot on the anode, which, although not essential, does seem to indicate increased radiographic value. In order to get high reading on the milliammeter with this tube, when used on coil giving spark as illustrated in Fig. 178, it is necessary to use at least 12 amperes on the primary; parallel spark gap resistance being 6 to 7 inches, and milliammeter reading between 6 and 8 milliamperes. The average exposure for adult hip, under such conditions, is from 20 to 40 seconds.

FIG. 4.—Illustrates a Tube that Shows an Inverse Discharge. Under such a condition the fluoroscope will be brilliantly illuminated, but the radiograph will be flat; that is, it will lack contrast between bone and tissue, and the definition will also be very poor. It is very important to note here that the milliammeter, when the tube is in this condition, will read zero; yet we may have exactly the same condition in the primary circuit as exists when the tube presents an appearance such as is illustrated in Fig. 3 of the present plate. The inverse discharge is so great that direct x-ray reading is completely wiped out. This inverse discharge may be overcome to quite an extent by using a series spark gap or ventral valve tube, by lowering the vacuum by means of the regulator and then raising self-induction of the primary. It is advisable not to operate the tube when it presents this condition, as the tube then becomes blackened and overheated. Overcome the tendency of the coil to produce this condition by the means suggested, viz., by the series spark gap or ventral valve tube. Some coils will operate much better if the spark gap is kept constantly in series with the tube.



H. F. AILKER DEL

STAGES IN "RIPENING" OR "SEASONING" X-RAY TUBES

and the discharge will jump across an air gap of 5 to 7 inches as estimated by means of parallel spark rods, with which nearly all coils are equipped. Under such conditions the tube should be lowered by means of the regulator, that is, by allowing the discharge to pass through the chemical or by heating the paladium regulator if of the osmosis variety. This allows gas to pass into the tube, and in this manner lowers the resistance. If the current is now turned on in sufficient quantity the tube will glow with an apple-green fluorescence. Bright fluorescent spots oftentimes appear on different parts of the tube; this appearance may last for several minutes if only a small amount of energy is allowed to pass through the coil. If, however, heavy discharges are sent through the coil, this condition soon changes in a new unseasoned tube with the Wehnelt interrupter, and a warning note is invariably heard just previous to this change, and the expert knows that the resistance of the tube is about to fall very rapidly unless the current is diminished or shut off completely. If the current is shut off just previous to this fall in resistance or lowering of vacuum, and the tube allowed to cool completely, this process may be repeated many times; but if the current is not shut off and the tube is allowed to run, the anode becomes hot, blue vapor appears in the bulb, the color changes to a more yellow-green, and then there is seen a stream of bluish vapor between the cathode and anode. At first, this stream impinges on the anode, but as the vacuum gets lower it will be seen that the cone-shaped stream changes and apparently two cones are formed having the apices midway between the anode and the cathode (Fig. 2, Plate V.). This blue stream is known as the cathode stream. Just previous to this double-cone appearance the anode becomes very hot. The radiographic value is greatly increased, then there is a sudden and greatly diminished radiographic value. The anode appears less hot, and finally the tube becomes completetv filled with blue vapor. During the latter part of this condition a stream of blue vapor may oftentimes be seen starting from the anode and apparently striking the glass either in a line perpendicular to the face of the anode or at right angles to the cathode stream. Where this blue stream strikes, the anode is said to be the source of x-ray light. Such a tube is of very little value for radiographic work, but if properly treated the process of seasoning will go on and eventually the tube will present an appearance very much like that shown in Fig. 3, Plate V. A beautiful rich yellow with a sharp line of demarcation divides the tube into two distinct parts; a brightly illuminated spot showing on the anode if the current is powerful enough. This bright spot is caused by the cathode stream striking the anode, and in a perfectly focussed tube covers an area of about one-sixteenth of an inch. If it is much larger than one-sixteenth of an inch in diameter, except under conditions described under low-vacuum tube (Fig. 2, Plate V.), it shows that the tube has not been accurately focussed, and radiographs taken with such tubes lack the fine definition so necessary to good plates. If the spot is smaller than about one-sixteenth of an inch the bombardment of the cathode stream has so much force that

the anode will be punctured if of light weight; if the anode is of a heavy type, as is the case in the tubes used by the writers, the face of the anode will be fused at this point, even with short exposure. When the tube reaches this stage (Fig. 3, Plate V.) it is of great value for radiographic work, although by no means fully seasoned. The process of seasoning, if properly managed, goes on from this point until we get a tube the color of which changes to that of a more yellowish-green resembling that of Fig. 1, Plate V., except that the color is richer and the fluorescent spots are not so persistent if at all present. When the tube reaches this last stage, which may take several weeks of daily use, it should be carefully tested and labelled, as it is now the tube needed for radiographs of the deeper parts. During this process of seasoning many interesting and remarkable phenomena may be observed. One of the most striking, after that demonstrated in Fig. 2, Plate V., is the condition of very high resistance which all tubes seem to pass through if kept in use long enough. The tube glows with this characteristically beautiful color when started up for a few seconds, then suddenly it seems as though a deep inspiration had occurred, for usually, without any warning whatsoever, the glow disappears and there is a loud roar in the interrupter; and even if more current is sent through the tube it cannot be started up again without lowering the vacuum. This is probably caused by the complete exhaustion of the terminal of occluded gases as well as by the using up of the original vacuum. When the tube reaches this condition it is necessary to lower the vacuum more frequently than before, by means of the chemical regulator. The tube seems to be able to take care of large quantities of liberated gases. It has been noticed by the writers oftentimes that the whole tube will be filled with a bluish vapor which will instantly disappear, and the beautiful rich greenish-yellow take its place. When the tube reaches this condition we consider it of the greatest radiographic value, it being capable, when properly operated, of giving good radiographs with very short exposure; also definition is greatly increased and there is very little diminution in the contrast so noticeable when using a yellow tube, as illustrated in Fig. 3, Plate V. To accomplish this process of seasoning and ripening an x-ray tube, weeks of careful manipulation during the daily routine work will be required, and it is possible to ruin all chances of ever bringing this condition about unless the operator appreciates certain basic principles in the proper manipulation of x-ray tubes. The temptation to beat all records for x-ray exposure is probably the cause of so many failures. To illustrate how necessary a sufficient length of exposure is, let us take the ordinary photographic process. Is it possible to get the best results of interiors or of subjects in which there is a variety of values as to light and shade by a short exposure? A short exposure of such a subject will usually produce a negative which shows under-exposure of the deeper shadows. We think the same holds true in taking radiographs of the deeper, thicker parts of the body and even of the extremities. We should remember that it takes time for the x-ray light to

pass through the part exposed to the photographic plate, and that the different parts of the body have different degrees of power to absorb light. If we can appreciate this important fact, our failures in this line of work will be fewer in number and our successes greater and more valuable.

Another condition, one of the most annoying and very common, is the tendency which some x-ray tubes have to be greatly influenced by the inverse discharge—an influence which is manifested by the appearance of several rings back of the anode, also throughout the entire tube. In addition to this the whole tube takes on the yellow fluorescence, and the line of demarcation so characteristic of tubes when operated to the best advantage is very faint. This condition may be overcome to a great extent by the use of the series spark gap so universally adopted. The spark gap used by the writers is the multiple spark gap introduced by Dr. Francis Williams, of Boston. Some coils are so constructed that this inverse discharge is great enough to jump several inches, as indicated by the fact that when this condition does occur in a tube it may be overcome by pulling the series spark gap out until the sharp anode line appears again. This condition of tube is illustrated in Fig. 4, Plate V. During this stage the fluoroscope is brilliantly illuminated, and it is possible to get radiographs of even the deeper parts with tubes in this condition, but they lack contrast and detail. The soft parts cannot be differentiated, and, except for the almost entire lack of detail, plates taken under such conditions, resemble those which are taken with the high vacuum tube. This condition usually occurs just before the vacuum drops, when the steady hum of the Wehnelt interrupter changes to the irregular roar. It may be overcome to a great extent by lengthening the series spark gap and turning off some of the current. If you are fortunate enough to have a coil with variable primary inductance, the difficulty may be almost completely eliminated by using a higher self-induction in the primary, two or more layers according to the degree of vacuum.

Following is a brief account of the important steps in the process of seasoning tubes as used by the writers. Before giving this account, however, we shall take the liberty of telling what we think to be some of the causes of so many tubes being ruined long before they have passed through the successive stages described above. The desire to get short exposure, and the lack of the appreciation of the fact that an x-ray tube is an exceedingly delicate piece of apparatus, are responsible for many failures. To connect up an x-ray tube, close the circuit, turn on the rheostat, and use the full capacity of the coil, particularly on a new tube in an endeavor to get a short exposure, is a serious mistake. You may succeed in getting a few good radiographs, but invariably the tube will go to pieces, blue vapor and the blue cone will appear, as illustrated in Fig. 2, Plate V., and, for the time being at least, the tube is useless.

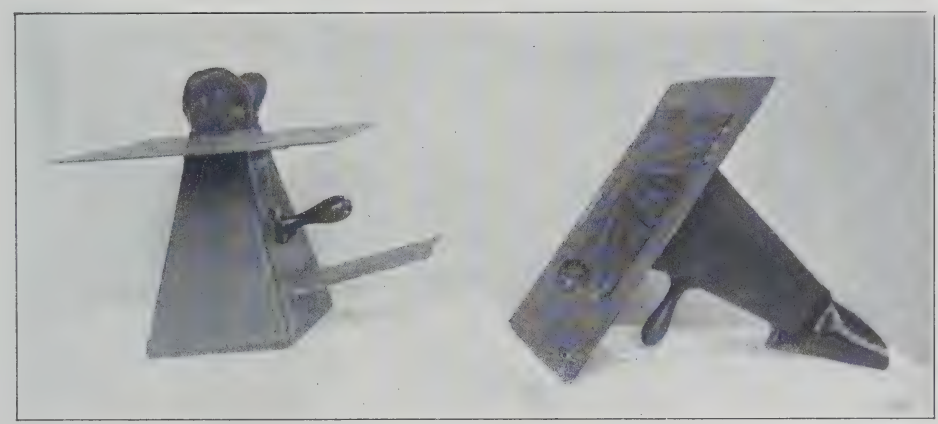
When a new tube is received it is tested by the following method: After being connected to the coil the current is turned on and the resistance of the

tube noted by means of the parallel spark gap or milliammeter. If the tube lights up with yellow color, as in Fig 3, Plate V., the current is turned off at once and the tube tested radiographically or by means of the milliammeter in series with the tube as described later. If the tube gives an appearance like that shown in Fig. 1, Plate V., more current is turned on; if fluorescent spots still persist and the color is green, the tube is lowered by means of a chemical or a palladium regulator. If a palladium regulator is used we employ an alcohol lamp with very small flame, the lamp being attached to a wooden stick four or five feet long, as advised by Dr. Rollins, of Boston. Palladium takes hydrogen from the flame, and this hydrogen passes into the tube and lowers the resistance. We consider this a most delicate and satisfactory regulator. After the resistance is lowered to such a degree that the tube has the appearance of that illustrated in Fig. 3, Plate V., it is tested radiographically or by means of the meter in secondary circuit. We have spoken of a stage in which the tube presents the appearance shown in Fig. 3, Plate V., but we should at the same time call attention to the fact that many tubes will resemble this in color, and yet be far from seasoned. We must say, however, that the tendency of the manufacturers to-day is to at least partially season tubes before shipping; this is done by running them on a coil during the process of exhaustion, and the practice is much to be commended. If the tube is of low resistance (see Fig. 2, Plate V.) the following method is adopted: The vacuum being low it is necessary to increase or raise it. If the coil has variable primary inductance, raise the self-induction of the primary. In many instances a tube may be made useful for radiographic work by this means. The series spark or ventral tube may be tried, but we believe it is better to begin the process of seasoning and proceed as follows: the tube is allowed to run with a small amount of current, only sufficient to bring the tube to a dull glow; this is to be continued for about twenty minutes, then more current is to be turned on until blue vapor just appears; the tube is now allowed to cool and the process repeated several times. If the operator is doing therapeutic work, this type of tube may be used to advantage on certain cases and the process of seasoning hastened in this way. After the tube has reached such a stage, resembling that of Fig. 3, Plate V., it is tested for radiographic value. We do not consider it fully seasoned when it presents the appearance shown in this figure, for this condition may be only temporary and the terminals and the other parts of the tube may still contain the gases that cause the vacuum to drop when overloaded or heated.

Radiographic Tests.

We have said that the tube should be tested radiographically, or by means of the milliammeter, in secondary circuit, with the tube. It will be noticed that fluoroscopic means of measuring x-ray light have not been referred to. For several years the fluoroscope has been completely dis-

carded by the writers except for the location of foreign bodies and in chest examinations. Many times during the earlier days of x-ray work it was observed that, while the fluoroscopic picture was exceedingly beautiful, the plates were of almost no value. After we had learned to judge the value of the tube by its appearance our results steadily improved. Having studied the fluoroscopic picture under all its varying changes, and having taken hundreds of plates during these changes, we feel compelled to say that this instrument is most unreliable. Even the ingenious devices, such as the skiameter, must be included in this sweeping statement. To say, when one is taking a radiograph of a suspected renal calculus in a patient weighing one hundred and fifty pounds, that the skiameter furnishes a certain reading, does not, as we believe, convey



A B

Fig. 183.—Two Fluoroscopes Used by the Writers for Different Purposes. (Original.)

A illustrates fluoroscope employed in making examinations of the chest or in searching for foreign bodies.

B represents an instrument formerly used when testing tubes. A skeleton hand is fastened on the bottom of the screen which is covered with black cloth. This instrument was discarded several years ago.

1 is a sheet of lead $\frac{1}{6}$ in. thick, painted with several coats of white lead paint; 2 is a sheet of aluminum $\frac{1}{8}$ in. thick and is large enough to protect the face of the operator; this is painted with several coats of white lead. Inside the top of the fluoroscope is set a piece of lead glass, $\frac{1}{4}$ in. thick, to protect the eyes.

information of value to anybody except the person who is conducting the operation, and even he will be in error sometimes. The fluorescent screen is deteriorating slowly but surely; the operator can at certain times make a better observation than at others, and what is to one observer a brilliant screen with black or gray bones or clear areas in the skiameter, may not be so to others. Then the all-important factor of the distance of the screen from the tube is oftentimes forgotten or neglected. We must remember that the value, both fluoroscopically and radiographically, increases inversely as the square of the distance. One of the very important objections to the use of the fluoroscope for such purposes is the danger to the operator. Fig. 183, A, illustrates an instrument that has been in use for several years by the writers; it is described as a protective

fluoroscope, and we still continue to use it when we have need of making such examinations.

The method used for testing tubes is the following: After trying a tube in the manner described above, apply the radiographic test. This consists in taking a radiograph of some subject that resembles, at least in its chemical structure, the human body. For this purpose the hind quarter of a good-sized sheep is often employed; this may be so cut that only enough to cover a 6½ by

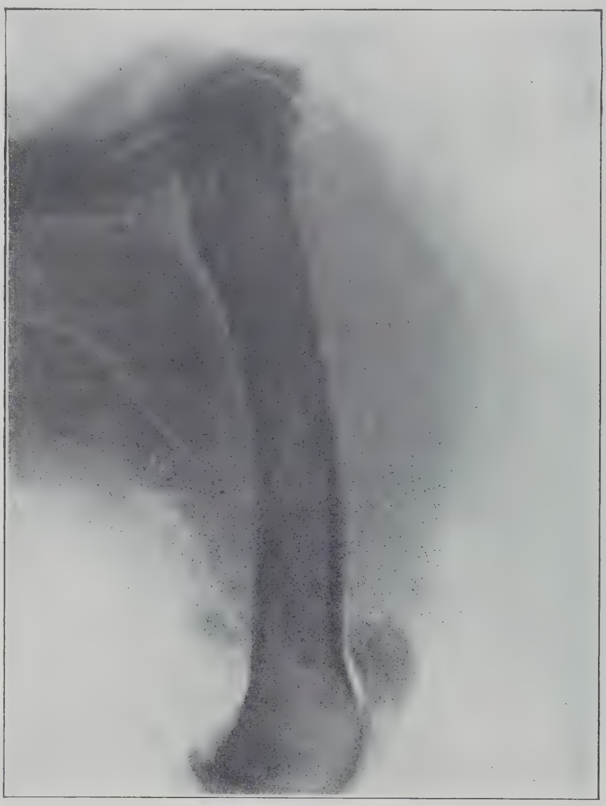


Fig. 184.—Illustrates specimen used when testing tubes. Exposure 10 seconds, distance 20 inches. Milliammeter reading 4 milliamperes; ammeter in primary circuit reading 12 amperes; spark resistance 6 inches. Under such conditions an adult hip would be given an exposure of from 20 to 40 seconds, according to size of patient; the tube being at the same distance from the plate. (Original.)

8½-inch plate is used. (Fig. 184.) This is then placed in ten-per-cent formalin for about ten days. It may be used fresh, and it is possibly better when it is in this state; but for the operator who has to test tubes quite frequently, the formalized specimen is more practical. After it has been removed from the formalin solution it should be washed and then placed in normal salt solution to which chlorinated soda in the proportion of 1:50 has been added. This is to overcome to some extent the objection that a dry specimen does not present the same conditions as the living. The writers are in the habit of using a dry speci-

men, one that was placed fresh in a formalin solution and allowed to remain there for thirty days; it is well preserved and in no way offensive. When this specimen is employed as a test, a piece of parchment paper is placed over the plate holder and an exposure is made. If the tube will take a thoroughly satisfactory radiograph of this specimen in from five to ten seconds it may be counted upon to take a good radiograph of an adult hip (160-pound patient) in two or three times that length of time; that is, in from ten to thirty seconds. We realize that this test is of a rather crude character, for in the living subject the

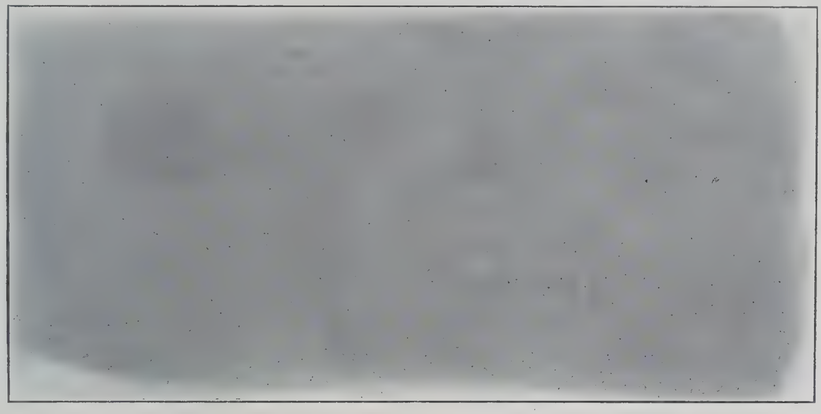


Fig. 185.—Radiograph Showing Renal Calculi; specimen consists of a block of beef six inches thick, three inches wide, and six inches long; kept under conditions described on page 614. (Original.)

physical conditions—the movements caused by the circulating blood, the amount of fat or muscular tissue clothing the part, etc.—vary enormously. Nevertheless, experience has taught us that we may rely upon it with some degree of confidence. For abdominal work—as, for example, in the search for calculi—the writers use a piece of beef about six inches square; on the top of this are placed capsules of gelatin which have been treated with formaldehyde, thus rendering them insoluble; these capsules, which vary in size from a weight of 1 grain to one of 15 grains, contain mixtures of calcic phosphate, calcic oxalate, sodium urate, and calcic carbonate. (Fig. 185.) The test can be made still more perfect by introducing, along with the capsules, a thin rubber sac containing partly air and partly water; the object of this step being to reproduce in some degree the conditions which actually exist in the living subject. The time of exposure required for securing a satisfactory radiograph under the conditions of this test is just about one-half that which will be required in the case of a living human being. The employment of such a test will generally save the operator from the necessity of making a second appointment with the patient.

Milliammeter.

Recently there has been perfected an instrument known as the ammeter for x-ray tubes; we refer, not to the hot-wire meter, but to the one invented by Dr. Snooks, of Philadelphia. The writers have done much work with this instrument and offer here their results. We believe it possible to utilize this instrument in estimating the radiographic value of x-ray tubes. This meter measures the amount of current passing into the tube, indicates a rise or fall in vacuum very accurately, and warns the operator at once of the presence of that harmful and annoying condition known as the "inverse discharge." To

Fig. 186.—Shows Increased Radiographic Value when Milliammeter reads Higher. Meter reading was 6 milliamperes, the other conditions being exactly the same as in Fig. 187. Increased radiographic value indicated by almost complete absence of soft parts. This effect could not be accomplished in the process of printing, and is due solely to increased value of x-ray light, as indicated by higher reading on milliammeter. (Original.)

illustrate this test we will take an apparatus giving a 10-inch very heavy flaming spark with ammeter on the primary circuit reading 12 amperes, tube 20 inches from the plate. A new tube is used; it is found to be of high resistance, the meter will not register over 1 milliampere, it has a parallel spark resistance of 6 inches or more. The vacuum is now lowered. (A Gundelach tube is preferred for this test, as the regulation of its vacuum is much more delicate and the vacuum obtained more permanent.) The ammeter will give a higher reading at once. If, however, the current is allowed to run through the tube for a minute or two it will be noticed that the indicator begins to swing back

and forth, sometimes going to zero; the tube during this stage presents the appearance of Fig. 4, Plate V. This swinging of the indicator is caused by the "inverse discharge," and on many coils it may be overcome completely by the use of the series spark gap, by the ventral tube, or by means of the variable primary inductance. If the current, however, is allowed to run on, in a few seconds the picture will change; the tube seems to quiet down, and the note in the interrupter is no longer intermittent and unsteady, but gives a loud roar. The tube is falling in vacuum; it becomes blue, and the cathode stream is seen. At *this point* it is interesting to note that the *milliammeter will show*

FIG. 187.—This radiograph was made with the milliammeter reading 2 milliamperes, the anode 24 inches from plate, and the time of exposure 10 seconds; developed 12 minutes. Compare with Fig. 186, which was taken under the same conditions exactly, except as regards the reading of the milliammeter, which was 6 milliamperes. (Original.)

its highest rating. We wish to emphasize the fact that the tube has the same appearance as that presented in Fig. 2, Plate V.—a very low tube, unfit for radiographic work, and yet the reading of the ammeter indicates its highest value. It is at this point that the amperage on the *primary circuit* and the parallel spark gap should be observed; it will be found that the meter in the secondary circuit reads *high,* while that in the primary circuit has a *low reading;* also that the parallel spark resistance has been reduced several inches, *i.e.,* from 6 or 7 to 4½ or 5. This condition will always take place in a tube that is *unseasoned* or in one that has been overloaded. *This high reading of the milliammeter does not indicate high radiographic value;* that is, if we mean by

this expression (x-ray value) the property of affecting photographic plates. Fig. 2, Plate V., illustrates this condition of low vacuum tube, giving high reading on milliammeter. *When a seasoned tube is used, and the spark resistance and amperage on the primary are carefully observed, the milliammeter will be found to be of great value.*

Fig. 188.

Fig. 189.

Figs. 188 and 189 represent radiographs taken on two different coils under exactly the same conditions of distance and time, with milliammeter reading the same in both exposures. Notice that radiographic values are equal. The same subject is used in Figs. 188, 189, 190, and 191. (Original.) Observe that these plates were taken on *different coils*, but with *same meter reading*. Other conditions were exactly the same.

Fig. 190.

Fig. 191.

Fig. 190 illustrates high radiographic value when milliammeter shows high reading—5 seconds; 6 milliamperes. (Original.)

Fig. 191 illustrates low radiographic value when milliammeter shows correspondingly low reading —5 seconds; 1 milliampere. (Original.)

The operator is invariably warned of this condition by the swinging of the indicator and the sound of the interrupter just previous to lowering of the vacuum. At this point it may be well to state that by having sufficient self-induction in the primary, a properly balanced coil, and service derived from a direct current of from 60 to 110 volts, this "inverse discharge" may be almost completely overcome.

As before stated, this unseasoned tube must be worked up in resistance. Label the tube and treat it as suggested. To avoid this low vacuum it is necessary that we do not overload the tube by crowding on the current or running it for a long time. When heating the palladium we should stop the heating process when the milliammeter reads two milliamperes. We may have used thus far only a part of the full capacity of the coil; if now more current is turned on, the meter may read a little higher or remain at two. Good radiographs of even the deeper structures may be taken with this apparently low reading if a seasoned tube is used. The process of seasoning is greatly lengthened by the use of tubes such as those of the Gundelach, Voltholm, and Friedlander type, owing to the large amount of metal in the anticathode or anode. Our observations have led us to firmly believe that the milliammeter may be used as an indicator of the radiographic value if, let us again emphasize *the phrase, "a seasoned tube"* is used. If the tube starts up with the appearance of Fig. 1 or 2, Plate V., it is of little value. With Fig. 1, Plate V., the reading will be low, $\frac{1}{2}$ to 1 milliampere: the radiographic value will be fair, the penetration high, the definition good, but the degree of contrast low. If the tube gives the appearance which is seen in Fig. 3, Plate V., the reading will be higher, the penetration good, the definition excellent, and the contrast very high. We have demonstrated this many times and have adopted this means of estimation, confirming it always by taking a radiograph of the specimen. (See Figs. 184 and 185.) The tube is then labelled with primary and secondary ammeter reading and length of spark resistance, which resistance we do not consider absolute, as so many conditions affect it. Figs. 188–191 illustrate the value of this method for estimating the radiographic value of x-ray tubes.

Development of Plates.

One of the reasons for poor results is failure properly to develop the plates. It is not necessary that the operator should be an expert photographer in order to get good radiographs, and the practice of sending x-ray plates to the professional photographer to be developed is to be condemned. The writers' experience has led them to adopt certain routine methods of development with standard formulæ and usually standard time, so that a man of little experience in this line of work can be sent into the dark-room with a strong likelihood that he will invariably get good results. The professional photographer usually underdevelops x-ray plates, and hence underdevelopment is one of the

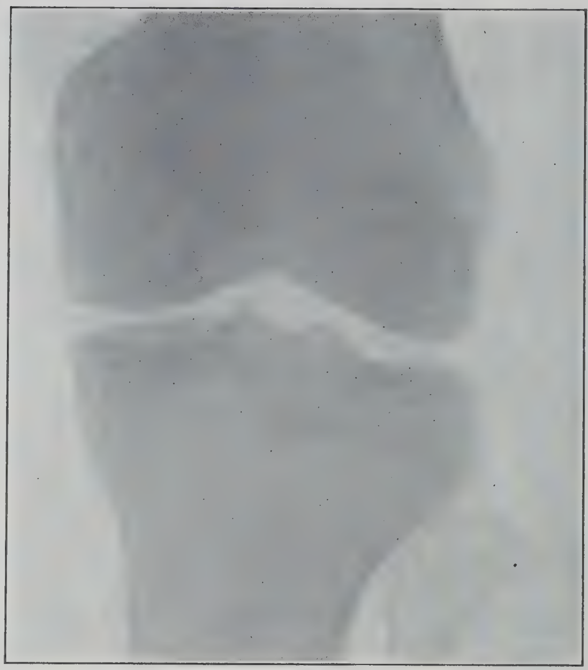


Fig. 192.—To illustrate undeveloped plate (only 6 minutes). Compare with Fig. 193, which was developed 6 minutes longer. (Original.)

Fig. 193.—Taken under exactly same conditions as Fig. 192, but development was carried much further. Developed 12 minutes. (Original.)

the great causes of poor plates (Figs. 185–192). It is of advantage to know the principles of photography, to know that the plate is coated with gelatin, that the agent acted upon by the light or x-ray is principally silver bromide, and that the richer the plate is in this silver salt the better the plate will be, other things being equal. For this reason, although ordinary photographic plates may be good enough for fractures and gross lesions, for the finer work they are not to be compared with the special x-ray plates (see illustrations Figs. 194 and 195).

Description of Dark-room.

A well-ventilated room from which all white light can be excluded is necessary. A single ray of white light may be responsible for poor results by acting on the photographic plate during the process of development. After a room

Fig. 194.

Fig. 195.

Fig. 194.—Ordinary Photographic Plate. Observe difference between this and Fig. 195. (Original.)
Fig. 195.—Same subject as Fig. 194, taken and developed under exactly same conditions, but on a special x-ray plate. (Original.)

which can be darkened completely has been obtained, the next thing of importance is to secure a suitable light by which to develop (Fig. 196). The light advised by the makers of x-ray plates is a ruby lantern or box through which the light is transmitted by means of two layers of yellow fabric and one layer of ruby fabric such as may be obtained from any photographic-supply store. The light to be used in these lanterns may be a 16-candle-power incandescent light, gas, a kerosene lamp, or even a candle; a candle, however, gives such a dim light that only one sheet of yellow fabric is necessary. At the same time

it is not safe to expose photographic plates even to this light for a very long time. This is especially true as regards ortho-chromatic and x-ray plates. Some form of tray in which plates may be placed is now necessary. An apparatus has been devised which consists of two or three shelves, each shelf capable of holding a tray. These shelves are placed on a base enclosed in a cabinet from which all light may be excluded by means of a light-tight door. This base, by means of transmission shafts and excentric cams, is given a rotary

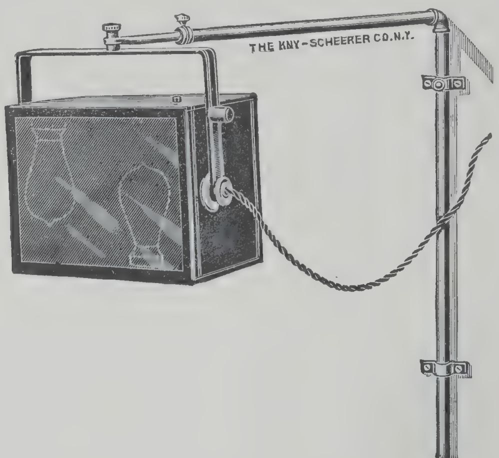


FIG. 196.—Dr. Caldwell's Dark-Room Ruby Lamp, with Bracket attached.

rocking motion when connected with a small electric or other motor. By this means the trays are kept in motion, not simply from side to side, but also in a rotary manner; in this way more even development is assured.

The following formulæ, which have been used for several years with very satisfactory results, may be confidently recommended:

Solution 1.		Solution 2.	
Sodium sulphate (dried)	432 grains	Sodium carbonate (dried)	735 grains
Hydrochinon	111 "	Water	16 ounces
Glycin	32 "		
Metol	5 "		
Potassic bromide	69 "		
Water (distilled)	ad 16 ounces		

Many other developers have been tried, but they seem to be less well adapted to this kind of work. It remains a fact, however, that after an operator has become accustomed to a certain developer he can get more satisfactory results with it than with any other. The developer recommended above will keep, and may be used over several times if poured back into the bottle, which should be corked tightly.

For the development of plates in which it is desired to show only the bone structure, ten parts of solution No. 1 to six of solution No. 2 should be taken. After these solutions have been mixed, the mixture should be poured into the

tray, in which the plate may then be placed. Great care should be taken to make the developer flow evenly and completely over the plate; if this be not done the plate will be overdeveloped in the parts that the solution first acted

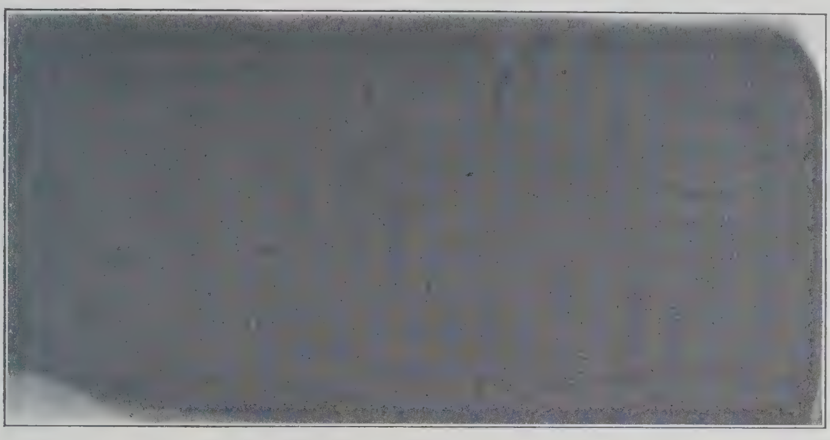


FIG. 197.—Illustrates underdeveloped plate of renal calculus. (The same radiograph as that shown on p. 615). Negative so thin that even under diffused light calculi cannot be seen. (Original.)

upon as compared with the rest of the plate. For this reason many operators advise the use of slow dilute developers, thereby avoiding the tendency of the image to flash up, while the high lights become overdeveloped before the shadows

FIG. 198.—Illustrates normal development of the same subject as that shown in Fig. 197. (Original.)

can be fully brought out. Usually the image will appear in about fifteen seconds with this developer, and development may be continued for about six minutes.

It is advisable then to look at the plate by holding it up to the ruby light; it should be placed over a sink so that the developer will not drop on the developing bench. If the bones can be seen distinctly, showing quite light, develop-

ment has not been carried far enough and should be continued until, when the plate is held about six inches from ruby light, the bones can hardly be seen. The plate should next be rinsed in cold water. This is important. It not only checks further development, but aids the action of the fixing and hypo baths in which it must be now placed before it is exposed to white light. The hypo bath is usually acid in reaction. The developer is alkaline in reaction. If we put the plate into this acid bath with a quantity of alkaline developer on it we soon neutralize the acid and precipitate the alum, which in many baths is the hardening agent. The following hypo bath is strongly recommended by the writers, it having been given tests during hot weather under extremely harsh conditions:

Sodium hyposulphite	parts ii.
Distilled water	parts iii.

Add to this solution:

Sodium bisulphite, in the proportion of	grains 100 to the O i.

After the plate is fixed—that is, when white spots no longer appear when the plate is viewed from the reverse side—it should be allowed to remain in the fixing solution for fifteen minutes longer; this will harden the film and clear the plate. It should then be washed in running water for about twenty minutes, or, if running water is not at hand, it should be washed for thirty minutes in four or five changes of fresh water. The plate should then be put on a rack to dry where dust will not be blown on it. We strongly advise the use of an electric fan for drying plates. For developing plates of joints, the chest, and, above all, for that important part of radiography, the location of renal and urethral calculi, the following mixture is advised: 8 parts of solution No. 1, 6 parts of solution No. 2, and 8 parts of cold water.

To properly develop plates of suspected calculi is by no means easy, and great attention should be given to this process. We believe that it is possible to detect calculi of very small size even when they are composed principally of urates (pure uric-acid stones being rare). (Figs. 197 and 198.) If a dilute developer is employed the process of development requires a longer time, but the contrast between the softer tissues is emphasized and in most cases the outline of the kidney can be made out. The plate is allowed to develop for about ten minutes before an observation is made with the ruby light. During this time the rocker is covered and the ruby light excluded. This precaution is advisable as plates may be fogged by long exposure to ruby light. If the plate, when held before the ruby light, shows distinct outlines of the lumbar vertebræ, development is allowed to proceed for a few minutes longer or until the vertebræ appear hardly discernible. The plate is then fixed, washed, and dried as before. The temperature of the developer is of great importance. If it is too cold, that is, below 65° F., development proceeds very slowly; if above 72° F. the plate is likely to be flat and devoid of contrast, being what is technically termed fogged. Always work

with the developer at about the same temperature. Renew the light in the ruby lantern when it is below candle power; keep the developing room clean. Use distilled or rain water, when possible, for developer and use filters on the faucets which supply the water for washing the plates. Upright fixing baths and washing boxes are advised.

Method of Taking Plates.

After the tube has been adjusted the next step of importance is the proper arrangement of the patient; and here success or failure may occur. To ask a patient to keep still in an awkward or uncomfortable position is asking much under the best of circumstances; and when he is actually under examination,

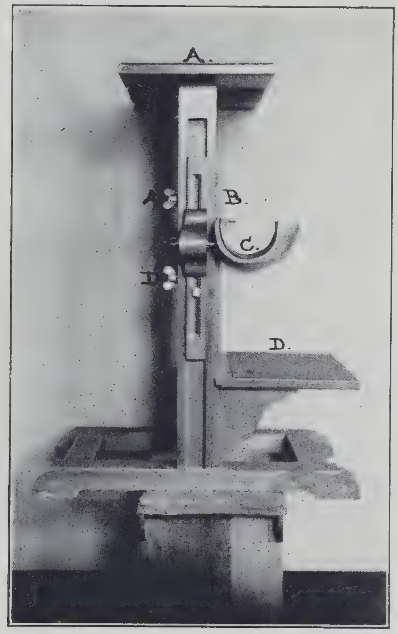


Fig. 199.—Table for standardizing position, avoiding the necessity of patient lying down for leg and ankle negatives and conducing to muscular rest and therefore quiet. (See also Figs. 200 and 201.)

A, Top adjustable by thumb screw, *A′*, for upper arm, elbow, forearm, and hand.

B, Rack for plate in lateral views of thigh, knee, and ankle. Clutch for holding plate seen directly below letter *B*. Rack attached to *D*.

C, Rest for thigh and ankle in lateral views of thigh, knee, and lower leg. Adjustable at thumb screw *c′*, Fig. 200. Tube placed in these views horizontally at same level as part, while plate is perpendicularly held on rack *B*.

D, Platform, adjustable by thumb screw D′, for antero-posterior views of thigh, knee, lower leg, and ankle. Plate rests flat on platform, which is raised to level of chair in which patient sits. Top *A* is lifted out of its sockets, and tube placed vertically above part.

D also is used for lateral position of ankle. (See Fig. 201.) A small table of thin wood, practically offering no resistance to the *x*-rays, is placed under the foot in the weight-bearing position. The plate is held behind it by clutch on rack *B*, and the tube placed horizontally is focussed over the malleoli.

Webbing straps and narrow sand-bags will be found useful in holding the parts immobile, and the ease and constancy with which standard views may be obtained are satisfactory.

Most of the principles here combined into one table were devised as separate apparatus by Dr. L. A. Weigel, of Rochester, N. Y.

surrounded by more or less apparatus the effect of which is unknown to him, it seems unreasonable to expect him to remain quiet. Many patients, particularly women, object to lying down, so that some form of apparatus is advisable which may be used when the arm, leg, or chest is to be radiographed. For this purpose the writers have devised the apparatus illustrated in Figs. 199 to 201. Having arranged the patient as comfortably as possible we must now place the plate beneath the part to be radiographed, and adjust the tube

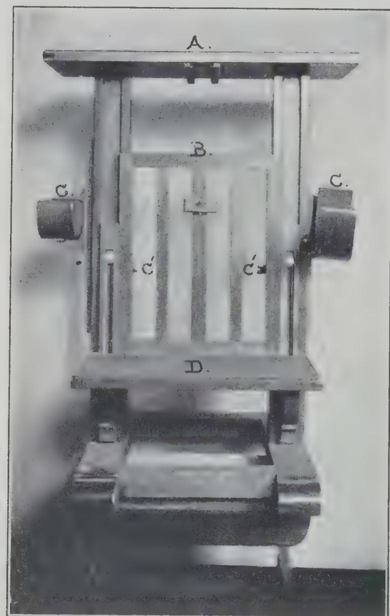


Fig. 200.—Front View of Table shown in Fig. 199.

at the standard distance for that part over the standard landmark, immobilizing the part as completely as possible by means of sand bags, straps, or, better still, by means of the method perfected by Dr. Albers Schoenberg; that is, by means of the compression cylinder to be described later. The writers have used for years several sizes of sand bags varying in weight from two to twenty pounds. These, if not filled too hard, may be used to hold even very sensitive parts almost perfectly quiet. It should be remembered that complete immobilization is absolutely essential to good radiographs. Simply asking a patient to keep still is not sufficient. Immobilize the part in every case no matter how simple.

We have said, place the tube at the standard distance for that part over the standard anatomical landmark. We feel so strongly convinced that this is essential to the intelligent interpretation of radiographs that the following scheme has been adopted and is in force at the laboratory of the Massachusetts General Hospital.

When taking radiographs involving joints the writers adjust the anode immediately over the centre of that particular joint, and take both antero-posterior and lateral views, great care being exercised to secure the same position as that of the standard radiograph with which it is to be compared. We fully realize that in many cases it is impossible, owing to ankylosis, deformity, or for some other reason, so to adjust the patient that this result shall be attained, but, wherever it is possible to do this, such procedure is insisted upon, and the uniformity in radiographs thus obtained warrants the extra time and trouble. To convince one's self of the need of such attention to detail it is only

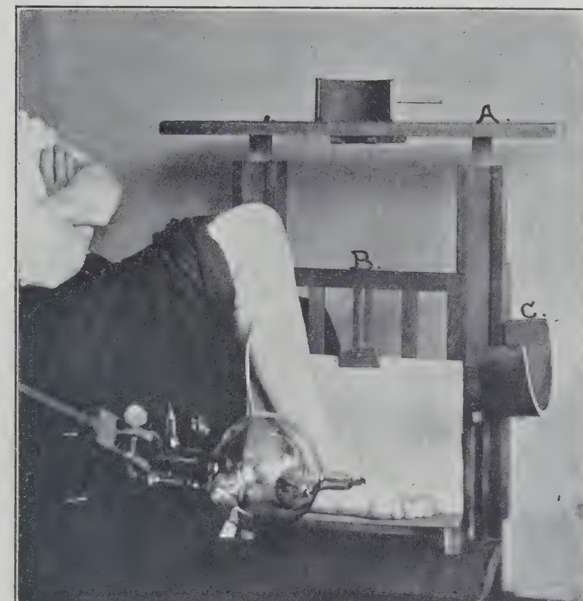


Fig. 201.—Table in Actual Use. (See Figs. 199 and 200.)

necessary to take radiographs of the head of the humerus. For example, one plate may be taken with the arm in outward rotation, and then a second one with the arm in inward rotation. A comparison of these two radiographs will show decided differences. In fact, many very different radiographs may be obtained of the same part by making comparatively slight variations in the position. For this reason some standard position and distance of the tube, and, whenever possible, standard position of the part, are essential. If an antero-posterior view is necessary, let it be such in reality, and not a three-quarter view, or one in which the plate was tilted a trifle, thus rendering comparison with the standard almost useless.

If the hip joint is to be radiographed, the anode is focussed over the joint as nearly as possible, the greater trochanter and the anterior superior spine being used as landmarks. The writers always work with the anode at least eighteen

inches from the plate; if the patient is a large, fat, or muscular individual the distance will necessarily be just so much greater—from two to four inches more. The greater the distance the less the distortion.

When taking radiographs for the localization of calculi, renal or ureteral, it is necessary to adopt the following routine procedure. The patient's bowels are completely evacuated, by means of Epsom salts and castor oil, twenty-four hours before the radiograph is to be taken; the patient is also requested to eat sparingly and only easily digested food during this time. This is considered

Fig. 202.

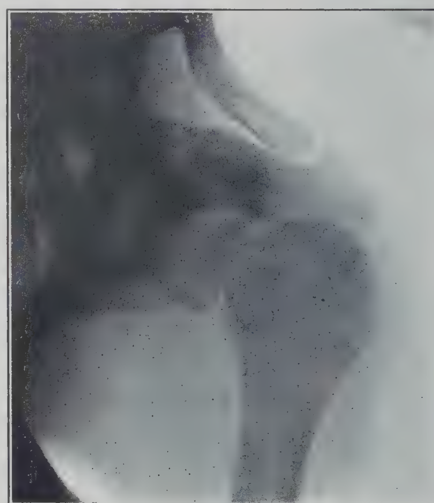


Fig. 203.

Fig. 202 illustrates the necessity of having the tube in a definite position. Notice the difference between this picture and Fig. 203. (Original.)

Fig. 203.—Same subject as Fig. 202. Position of tube was changed and patient's arm rotated. (Original.) Notice difference in width of head of humerus as compared to Fig. 202.

of such importance that it should be insisted upon. When the patient is ready to be radiographed, he is placed on the table, with the shoulders elevated and the legs flexed (see Fig. 204). The compression cylinder is then adjusted, so that the upper border of the cylinder is just level with the seventh or the eighth costal cartilage. The cylinder is next pressed down firmly, then tilted upward; by which means the last two ribs will be made to appear on the plate, thus enabling the operator to be sure that the entire kidney is under observation. It is always advisable to take plates of both kidneys and of the entire ureteral tract. This makes it necessary to take several plates; but, as we feel convinced that nearly all calculi may be detected by means of the x-ray (except, possibly, uric-acid), such procedure should always be observed in cases of suspected calculi. Great care must be observed in the interpretation of such radiographs. The illuminating lantern (Fig. 176) should be used and the plate carefully studied. The plate should show the last two ribs, the transverse processes of the lumbar

vertebræ, the psoas magnus, and the quadratus lumborum muscles; and in patients of one hundred and seventy pounds or less the outline of the kidney should be made out. Under such conditions we believe it possible to detect very small calculi even when they are composed principally of urates.

It should be remembered that sesamoid bones, vein stones, also cheesy

FIG. 204.—Shows how cylinder may be used with ordinary rattan couch. Care must be taken to fasten the cylinder so securely that respiratory movement will not move cylinder. This is necessary, as cylinder and tube holder are attached to the same support, and consequently any movement of the cylinder will move the tube and thus spoil definition. (Original.) Apparatus adjusted for suspected renal calculi.

deposits in the calyces of the kidneys have been mistaken for calculi; and we believe also that in one case a foreign body in the appendix was mistaken for a ureteral calculus.

LOCALIZATION OF FOREIGN BODIES.

For the localization of foreign bodies the following method may be adopted:—

Whenever it is possible to do so, the foreign body should be located first by means of the fluoroscope. The fluoroscope used by the writers is illustrated

in Fig. 183, *A*. A diaphragm is used in connection with the tube so that only a limited field is under observation at one time; by this means the fluoroscopic localization is rendered much more accurate, as a foreign body, if one should be observed, must necessarily be confined to the area of illumination, such

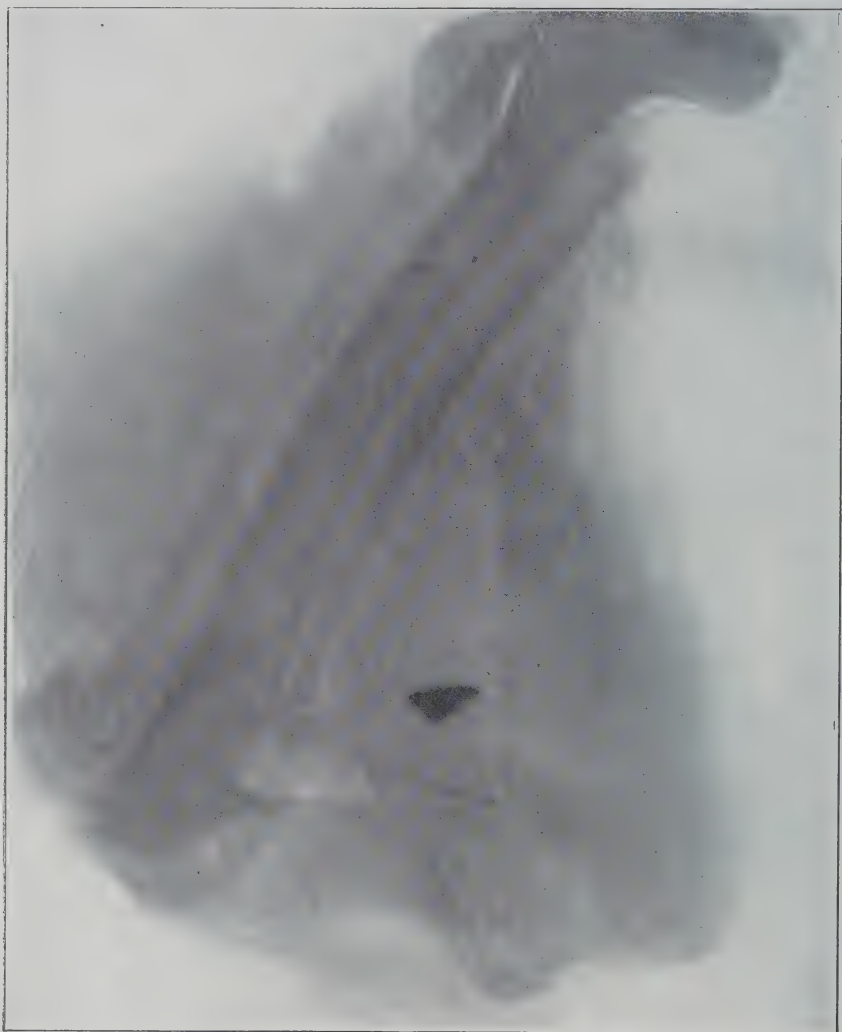


Fig. 205.—Represents specimen with needle buried in it. Dark triangular shadow near needle is a piece of lead used as a mark (the same as that used on patient's skin). Notice the two shadows of the needle. Anode was $16\frac{3}{4}$ inches from plate. Shadows of needle exactly $\frac{1}{4}$ inch apart. Tube was moved 3 inches to the right, then 3 inches from the left, of central point. Needle was $\frac{7}{16}$ of an inch deep. (Original.)

area being dependent upon the size of the diaphragm. (A one-inch diaphragm, at a distance of about twenty-four inches from the tube, gives a three-inch field.) The orthodiagraph, as devised by Dr. Moritz, is highly recommended for this purpose. After the foreign body has been located in one plane by means

of the fluoroscope, such location should be marked on the skin of the patient. The plate is next to be placed in position. The anode of the x-ray tube is then focussed over the mark made on the skin. The distance of the anode from the plate is carefully measured and recorded. The writers work with the anode at a distance of fourteen inches from the plate. After the tube has been accurately focussed over the mark on the skin, which corresponds to the foreign body, the tube is to be moved three inches to the left and an exposure made. The tube is then to be moved first back to the central or starting-point and then afterward three inches to the right. Great care shoud be used in making the measurements, and the exposures must be made at the same distance (fourteen inches) from the plate. The plate is now to be developed, and, unless the object is located at a considerable depth from the skin, two images will appear. The distance between the two images is measured and the depth calculated (see Figs. 205 and 206).

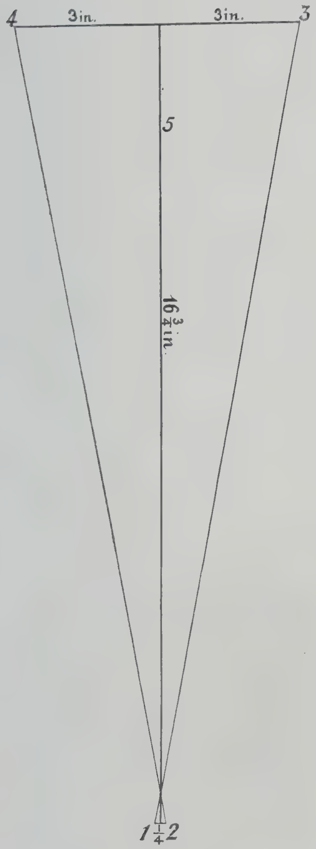


FIG. 206.—Illustrates plan of calculation as to location of foreign body.

Nos. 1 and 2 correspond to shadow of needle, and are ¼ inch apart—the same distance as the shadows on the plate. Nos. 3 and 4 correspond to the two positions of the tube. No. 5 corresponds to distance of anode from plate. The point where the lines intersect represents depth of body from surface next to plate. (Original.)

Many operators use two separate plates rather than make two exposures on one plate. This may be necessary in some cases, but, as a rule, one plate with double exposure will do. The writers always fasten a piece of lead on the patient's skin somewhere near the foreign body. This object appears on the plate and is a guide to the surgeon. Dr. MacKenzie Davidson has devised a very ingenious method and apparatus for the localization of foreign bodies.[1]

For locating foreign bodies in the eye the apparatus and technique of Dr. Sweet, of Philadelphia,[2] may be used to advantage.

[1] Lancet, 1897, p. 1001.
[2] Archives of Ophthalmology, 1898, p. 377.

Compression Apparatus.

To omit to mention in the description of an x-ray plant the compression cylinder of Albers Schoenberg would be to omit one of the essentials to good and accurate radiography.

This ingenious device is so thoroughly described and illustrated in the catalogues of dealers in x-ray apparatus that only the principles and advantages need to be spoken of here. At the same time it will not be out of place to give here a brief description of the compression cylinders which we are in the habit

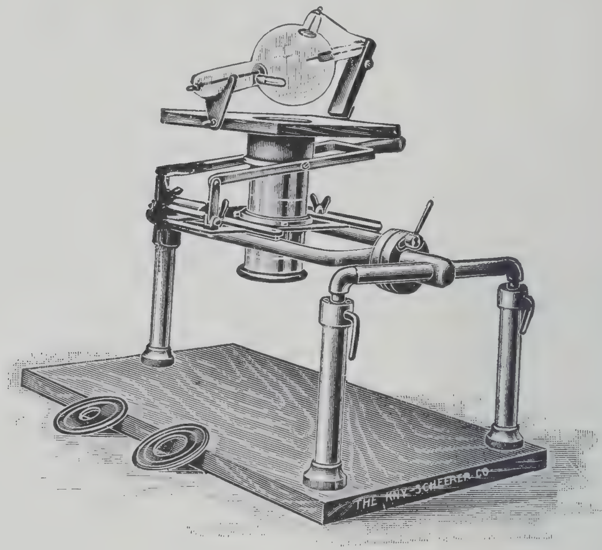


Fig. 207.—Compression Diaphragm of Albers Schoenberg. It consists of an adjustable frame on a wooden base, with a detachable lead-lined compression cylinder (4 inches in diameter), lever arrangement, tube-holder, and three lead diaphragms.

of using, these cylinders being modifications of Albers Schoenberg's much more elaborate apparatus. (Fig. 207.)

On a previous page we have spoken of the absolute need of immobilization of the part to be radiographed. The compression cylinder furnishes the ideal method of immobilization. By this means the part may be completely fixed and all movement prevented, thus obviating one of the chief causes of poor plates.

Secondary radiations have been spoken of as being another cause of the lack of definition, of fogged or blurred plates, such radiations being given off to some extent by all objects with which the x-ray comes in contact. By means of the compression cylinder with its diaphragms, secondary radiations are almost completely eliminated, only the more direct rays being used. (Figs. 211 and 212.)

The importance of focussing the anode over the part to be radiographed has been emphasized. The compression cylinder enables the operator to do

this very accurately, as the centre of the cylinder corresponds to the focal spot on the anode of the x-ray tube. Its advantages are: uniformity of distance of the anode from the surface of the body, thus setting a standard distance; greatly increased definition owing to elimination of secondary radiations; better immobilization of the part exposed; and, finally, the fact that in the more compressible parts of the body, such as the abdomen, the cylinder enables the operator to focus the tube over the area to be observed as well as to compress the part and thus reduce its thickness to a considerable degree. Another

FIG. 208.—Shows how tube-stand table is used when antero-posterior view of knee is desired. Patient is in chair, and leg is placed on adjustable table, as described under compression cylinder. (Original.) The operator may be observed behind the lead screen in which a hole 4 inches in diameter is cut. This hole is covered with lead glass $\frac{1}{4}$ inch thick.

advantage, and one that will appeal to the operator who has to use many plates, is the fact that with this apparatus only those which measure either five by seven or eight by ten inches can be employed. The cheapness and efficiency of the apparatus are features which also commend it to favorable consideration.

It is constructed in the following manner: A board three-fourths inch in thickness, fourteen inches long, twelve inches wide is taken. In the centre of

this board a hole three and three-fourths inches in diameter is cut, with a groove one-fourth inch wide, three-sixteenths inch deep. Into this hole is set the diaphragm. The diaphragm consists of a piece of lead three-sixteenths of an inch thick, in the centre of which a circular hole one and three-fourths inch is cut. On the bottom of the board a sheet of lead three-sixteenths of an inch thick is fastened so that it covers the board to within one-half of an inch of

Fig. 209.—Shows correct adjustment of tube to diaphragm. Elliptical shadow (see Fig. 210) is caused, probably, by secondary radiations, and indicates the value of diaphragms in radiographic work. A fluoroscope will give almost equal illumination when held 18 inches from tube, it being thus impossible to detect by this means the secondary radiations. (Original.)

the edges. Three cleats are fastened to the lead and are so arranged around the circular aperture that the cylinder, about to be described, may be slid in or out, according as the diaphragm is to be used with cylinder or not. One cleat is fastened near the back of the aperture so that the centre of the cylinder, when pushed in against it, must be exactly under the centre of the diaphragm; the other two cleats are fastened so as to centre the cylinder in the lateral direc-

tion. The cylinder used by the writers consists of a tin can, such as compressed tablets come in; this is lined with sheet lead one-eighth of an inch thick and soldered so as to be securely held to the sides of the can. Two lugs, or ears, are now fastened to the sides of the upper end of the can, these lugs fitting into the cleats on the board as described. To the top of the board are fastened

FIG. 210.—Shows how necessary it is accurately to adjust the tube to the diaphragm; care being taken to place foca spot of cathode stream immediately over centre of diaphragm. This may be easily accomplished by placing fluorescent screen under cylinder. If complete circle with sharp, well-defined edges is seen, adjustment is correct; if illumination is elliptical, as in this figure, the tube must be moved either to the right or to the left. (Original.) Notice dark shadow inside lower part of circle, obscuring part of ulna and radius. Compare with Fig. 209.

two uprights for supporting the x-ray tube; these uprights are five inches high, the ends of the uprights being grooved to receive the x-ray tube.

This cylinder, with board and tube holder, is now fastened to a very rigid upright in such a way that it can be raised or lowered at any angle in the same manner that the ordinary tube stand allows adjustment of the x-ray tube. Absolute rigidity of the upright is essential, as, when a radiograph of the lumbar

spine, for instance, is to be taken, the cylinder is pressed down as much as the patient can stand, then fastened in front by means of a strap passing through the board to the couch (see Fig. 204). If the upright is not stiff, or if there is any play in the board arm, respiratory movement will move the apparatus which, moving the tube, will spoil the definition and thus defeat the chief aim of the compression cylinder.

Figs. 204 and 207 illustrate this apparatus, with adjustable table on the same upright.

The writers consider the compression cylinder essential to good radiographic work. The apparatus described has the advantages of being easily and quickly adjusted and purchasable at a reasonably low price. The cylinder being movable enables the operator to use the board alone when larger areas than that allowed by the cylinder are to be exposed. In such cases straps and sand bags are used when possible to immobilize the parts.

Stereoscopic Radiographs.

The use of the stereoscope is of undoubted value in the study of the position of bone fragments and foreign bodies. It necessitates some form of stereoscope, and the taking of two negatives under exactly the same conditions as regards time of exposure and mode of development, and with the tube at the same vertical distance from the plate in each case. After centring the tube over the point chosen as the most desired area to be viewed, the tube, by means of a scale on the tube stand or floor, should be moved horizontally one-half inch to the right and a negative taken. This plate should then be removed and a second one substituted, with no change in the position of the part. The tube should next be moved three-fourths inch to the left or one-half inch to the left of the original central point, and the second negative should then be taken under the same conditions as regards distance and light overhead. Thus we shall obtain two negatives which give us views of the area desired from two different positions corresponding roughly to the human pupils. It remains for the stereoscope to construct the images in three dimensions.

Harmful Effects of the *X*-Ray.

Nearly all the measures used for the relief of suffering are capable, when used ignorantly or carelessly, of producing in time untoward effects. The *x*-ray is no exception to this axiom, and the lesions which follow its use are among the most insidious and the most disastrous. Our present knowledge of these matters is most incomplete. Enough, however, has been already proved to warrant very definite statements, and the evidence as to other possible harmful effects is sufficiently conclusive to demand the adoption of protective measures which at first sight may seem unnecessarily strenuous.

It was early recognized that the x-ray exerted a very definite effect upon diseased tissue, and that long or repeated exposures occasionally produced a reddening of the skin or even a deep slough, the onset of which was slow and the result of which was a tissue necrosis most obstinate in healing. This gradual appearance of the lesion many days after the patient's exposure, and the discovery made by many operators that the frequent short exposures to which they were constantly subjected were causing lesions on their own persons, demonstrated the subtle cumulative effect of this new and mysterious ray.

The difficulty of determining the danger point of these exposures is very great, for there are usually scarcely any subjective symptoms at the time when the patient is exposed to the ray. The martyrdom of those men who began their x-ray investigations soon after its discovery has been a very real one. The lesions which have resulted have been in many cases ineffaceable and have entailed an immense amount of physical and mental suffering.

The danger is a positive one, and we are doing our duty neither to ourselves nor to our patients if we do not adopt measures which are known to be adequately protective.

The x-rays derived from a static machine are somewhat less likely to produce untoward effects than those from the more powerful currents of the modern coil. But even the static rays are by no means innocuous. The experience of some of the most brilliant operators has shown that extremely painful lesions may be produced by this form of current, and a malignant growth may ensue in the ulcerations.

Sterility.—The investigations of Dr. F. Tilden Brown, of New York, and others have seemed to show conclusively that long-continued exposure to the rays may produce at least a temporary sterility. In some cases the spermatozoa are rendered non-viable, and in others they entirely disappear from the semen.

Whether a sterility thus produced is ever permanent, provided further exposure ceases, is still in doubt. Such a permanent change in the glandular structures seems possible.

The striking absence of children in the families of x-ray workers who have been constantly exposed to the rays without adequate protection is noteworthy. The cumulative effect is here apparently very important.

Before this subject was much discussed Dr. W. L. Rollins, of Boston, called attention to the fact that the Roentgen rays have the power to cause abortion in guinea pigs. These experiments are suggestive of the necessity of exercising caution in exposing pregnant patients.

It is probable that the female organs of generation are affected in very much the same manner as are the male when exposed to the effects of the x-ray. Cases can be multiplied in which highly penetrating rays have been administered for comparatively long periods of time in the neighborhood of the

genitalia, and yet subsequently the patients have become mothers or fathers of healthy children. It seems reasonable, therefore, to conclude that single exposures of short duration do not as a rule produce permanent sterility in either sex.

Burns.—*X*-ray burns, so called, are of different degrees, from a simple erythema to a definite tissue necrosis which may involve the layers beneath the true skin. There is an individual idiosyncrasy in the matter of susceptibility, just as there is a great difference in the effects of sunlight on the skins of different persons.

There may be considerable delay in the onset of symptoms. The reported intervals of five and six months seem hardly conceivable, and yet the writer has on his wrist a typical scar from an *x*-ray burn of the first degree which did not appear until after the lapse of over six months from the time of exposure. It should be stated, furthermore, that the wrist had been, as was supposed, adequately protected, and that no stage of erythema had been observed. The changes in the subcutaneous vessels, with the resulting irregular red mottling of the skin, has persisted now for about two years.

Depilation may occur, after prolonged exposures, without definite burns. It will perhaps be noticed that many *x*-ray workers have lost eyebrows and eyelashes. In some cases also the nails become brittle and ridged. A troublesome seborrhœa is a not uncommon result, and small keratoses or areas of thickened epithelium, which present the appearance of small non-sensitive calluses, are often seen, especially on the hands.

Few lesions are so persistently painful or so slow in healing as the deep *x*-ray burns. Paradoxical as it seems, areas of malignant disease often occur in these sluggish granulations, and that, too, notwithstanding the fact that the original lesion, in these cases, owes its origin to the agent so much exploited in the cure of malignant growths.

Pathology of x-Ray Burns.—Vose and Howe have made careful studies in the effects of the Roentgen ray upon cancer (*Journal of Medical Research*, vol. xiii., No. 2). Their conclusions, from a study of the literature and from their own microscopic examination of tissues removed from these burned areas, are as follows: "Sections from the tissues of such burns studied by us show progressive changes from the surface downward, the more highly organized parts naturally showing the most marked changes or suffering most. The hair follicle and the glands are destroyed. The prickle-cell layer is increased. The cells of this layer show granular degeneration of protoplasm and proceed to necrosis. The blood cells show a reticular deposit of fibrin on their inner coats. No change of nerves was noted. These histological changes are all that may be positively claimed, since the sections of *x*-ray ulcers show a purely necrotic process—increase of elastic tissue, increase of connective tissue, and colloid replacement."

Protective Measures.

In the light of these facts efficient protection of both the patient and the operator has become a matter not only of wisdom, but also of imperative duty. Theoretically, as Dr. W. L. Rollins early pointed out, a box of sufficient *x*-ray density to cut off the probably harmful rays should completely surround the active tube, the only exit for the rays being furnished by a small fenestra through which the cone of light is directed against the desired part. Practically, with our increased speed of radiography and the more perfect technique which does away with repeated trials for the purpose of securing a good plate, we cannot feel that the risk to the patient from a single exposure to a naked tube is great. Additional knowledge may well change this opinion; and in any event the generative organs should always be protected by a piece of sheet lead.

As regards the operator no safeguards can be too complete. The *x*-ray atmosphere in which he works is in itself a baneful influence which it is impossible accurately to estimate.

Although in years past we felt it necessary to judge the actinic quality of the rays by fluoroscopic inspection, we have entirely given up this test, not only on account of its unreliability, but also because of its demonstrable danger. If this method is ever used by the beginner—and we discourage even this—the fluoroscope should be a protective one such as has been described elsewhere in this article. By careful observation of the appearance of the tube, its shade of color, the amount of current used, and especially by the reading of the milliammeter, we believe that even the beginner may learn, after a few simple trials on test plates, accurately to judge the quality of the light without the use of the fluoroscope. This inspection may be made through a peep-hole in the protective screen, which peep-hole should be covered by a thick layer of lead glass. For the fluoroscopic examination desirable in certain cases of foreign bodies or fractures, and in the case of thoracic or abdominal diseases, the observer should be guarded by a lead screen or a protective suit of the lead and rubber composition now on the market. Of the efficient protectiveness of this latter material we are not sure.

In our private office plant the arrangements are such that the current can be turned on only when the operator stands behind a permanently fixed screen, which is composed of sheet lead one-eighth of an inch thick and is placed between two layers of plate glass. A peep-hole one inch in diameter is cut in the lead. The screen is six feet high and five feet broad, and the lead is grounded by means of a wire attached to the gaspipe. In taking radiographs the long axis of the tube is placed at right angles to the screen, and the negative or cathode pole of the coil is farthest away from the operator, so that fewer rays are projected in his direction. The grounding of the lead screen, which collects the

high induction waves, ensures a safe disposition of this possibly harmful element. We do not consider these safeguards as unnecessarily extreme.

We are dealing with a force the exact nature of which we do not understand. It is more subtle, perhaps, than any other influence in the hands of medical men, partly because it is not yet fully understood, and partly because of its insidious workings. The harm it is capable of doing is second only to the good its application daily accomplishes, and the efforts of those who essay its use should be earnestly directed toward making it an unmixed blessing for both patient and physician.

Screen for Protecting the Operator.—The protection of the operator from the injurious effects of continued work in x-ray atmosphere is of the utmost importance, not only because of the liability to x-ray dermatitis, but also because of the possibility that the x-rays may induce sterility, as shown by Dr. F. Tilden Brown, of New York. Too much emphasis cannot be laid on this subject, and every means should be taken to avoid exposure. All fluoroscopic measures supposed to furnish information regarding the photographic value of x-ray light should be abandoned and the operator should learn to judge the value of the tube in use from the color of the fluorescence, from the resistance of the parallel spark gap, by means of the milliammeter in the secondary circuit, and by testing the tubes radiographically as described under x-ray tubes. The writers use, for the operator's protection, a lead screen (Fig. 208), three-sixteenths of an inch thick, in which a window is cut. In this window is set a piece of one-fourth-inch lead glass. The operator stands behind this screen, which is about six feet high and four feet wide. Through the window he observes the tube and in this manner is in all probability kept from harmful effects. When it is necessary for him to go near the machine during its operation he wears a lead apron, lead-filled gloves, both of which, as well as protective tube shields, are now on the market. When it is necessary to use the fluoroscope, as in chest observations and in locating foreign bodies, the protective fluoroscope is used as described (Fig. 183, A). In view of the observations made by Dr. F. Tilden Brown on the question of sterility it is advisable that the bodies of all patients be protected by means of flexible lead screens or protective tube shields. As the harmful effect of the rays is probably dependent upon direct and comparatively long exposures, we believe that the patients run no particular risk during the radiographic process. Nevertheless, in view of the lack of evidence on this point, it is advisable that the proper precautions should be taken.

II. THE INTERPRETATION OF RADIOGRAPHS.

In unskilled hands the therapeutic use of the x-ray includes the serious danger of burns. To the practitioner unfamiliar with normal x-ray anatomy, the interpretation of skiagraphs offers the no less serious danger of making an incorrect diagnosis and carrying out a vicious or useless treatment.

There are certain definite rudimentary conditions which must be complied with if our interpretations are to be of a sufficiently trustworthy character.

(1) It is preferable, whenever this can be done, to examine the plate itself rather than a print taken from it.

(2) In examining a plate two factors are of great importance—the management of the light, and careful attention to the distance (from the source of light) at which the plate is held. In the first place, the light should be evenly diffused, and at the same time it should be shut off in such a manner that it shall illuminate only the negative.

The matter of proper lighting, especially to one somewhat unfamiliar with the examination of negatives, is of sufficient importance to warrant the description of a practical illuminator (see Fig. 176).

An open square box, the sides of which measure four or five inches more than the edges of the largest x-ray plate likely to be used, is painted white inside, and the antero-posterior depth at the top is made slightly less than the antero-posterior depth at the bottom, so that a plate resting in a frame applied to the front of the box will be in no danger of falling outward, and yet its position will be nearly perpendicular. A removable frame having an open space still slightly larger than the largest plate is now fitted to the open box and held in place perhaps by hinges, hooks and eyes. On the inner side of this frame, above, below, and laterally, are fastened one or more candle-shaped incandescent bulbs lying flat along the side and having individual turn-off buttons, but wired to a common plug on the outside of the box. To the outside of this frame is fastened a revolving circle having a rabbeted opening of the exact dimensions of the largest plate to be used. Other rabbeted frames or kits, down to the size of the smallest plate, are now fitted accurately into this and into each other, and the illuminator is then ready for use. The bulbs throw their light against the white back and sides of the box and, as a result, an almost perfectly diffused illumination, which can be regulated in intensity by turning off some of the incandescent lights, or by means of a small rheostat on the outside of the box, is reflected through the opening. The advantage of the revolving circle is at once evident when it is considered that the standard sizes of plates are all longer in one diameter than in another. Thus, for example, a pelvis on a large plate is best viewed as if the patient were standing, and hence it is desirable that the longer of the two diameters of the plate should be the horizontal one; while in the case of a thigh the vertical diameter of the plate should be the longer one. The revolving circle obviates the necessity of constantly changing *en masse* the position of a rather bulky piece of apparatus. With the intensity of this evenly diffused light regulated according to the density of the negative, the best effect, except for the finest detail, is gained by studying the negatives at some distance from the illuminator. The less evident lesions and differences of shadow are thus much easier to appreciate. A method

warmly recommended by one of the best foreign interpreters is that of observing a negative, thus evenly illuminated, through a pair of opera glasses.

A good negative is something absolutely indispensable. By this is meant a negative in which soft-part details are not obliterated and in which fine bone structure is shown. It is possible by a proper choice of the quality of the light so to control the time of exposure and method of development that soft-part lesions will be most favorably shown; while with more penetrating rays, longer exposures, and with the development carried further, the bone structure may be emphasized. In the thinner parts a combination of both can usually be obtained.

Data of Position.—We have alluded above to the importance of standardizing our positions, and in the accurate interpretation of negatives this is of great moment. Before attempting to form any judgment we must at least have the data of position clearly in our mind. To appreciate possible distortion one has simply to place his arm between a fluoroscope and an active tube and then to move the fluoroscope and arm laterally. The amount of distortion thus to be observed is surprising.

Comparison with the Normal.—In the medical school the student has a long and thorough training in normal histology before he is shown pathological tissue. He must dissect normal subjects before he can be expected to recognize the gross lesions.

In skiagraphy we are dealing with shadows of structures, not with the visual and tactile examination of these subjects.

X-ray anatomy differs materially from that of the dissecting-room. We must learn to know the internal structure of the bones and must realize that we are looking through bodies of three dimensions and not at a single plane surface.

The bony structures of children at different ages differ very much from each other and from those of adult life. Yet the adult structures are the only ones in which we receive our anatomical training, while the bone lesions of children are more common and of greater import than those of adult life.

The ununited bone centres of the epiphyses have not seldom been spoken of as fractures, and the normal exostoses and bone ridges at the points of attachments of ligaments and muscles have been declared to be pathologic.

It must not be forgotten that sesamoid bones often develop in other tendons than the flexor longus hallucis and are usually of no significance to the possessor.

It is of importance, therefore, that we should become familiar with normal skiagraphs of all ages and compare either mentally or actually the radiograph supposed to be pathologic with a normal one of approximately the same age. Thus only can we progress in our power safely to use the Roentgen rays as an accurate method of diagnosis.

In the majority of cases the lesions are unilateral, and we have the other

side for our most perfect normal standard. Where it is not practicable to view both sides on the same plate at the same time, symmetrical positions should be separately taken. One acts as a check upon the other, and individual idiosyncrasy does not stand for the suspected lesion.

Fractures.

The diagnosis of fracture is usually the easiest of x-ray interpretations. It is extremely hard, however, accurately to determine, from an x-ray plate, how outwardly deforming even a marked solution of continuity may be, or to

Fig. 211.—Subperiosteal Fracture of Radius. Faint line of solution of continuity discernible. (Or ginal.)

predict how much disturbance of function the lesion is likely to cause. The necessity of having clear plates that show the bone structure well, is emphasized in the common subperiosteal solutions of continuity, in which the outline of the bone shows no irregularity (Fig. 211).

In all but the subperiosteal fractures it is advisable to take at least two

views of the injury; and careful data relating to position must be at hand if we are to interpret the plate correctly (Figs. 212 and 213).

From a medico-legal point of view the x-ray evidence of the presence of a fracture must needs be conclusive, but any inference as to the future disturbance of function that is likely to result from a seeming malposition should be drawn with the greatest caution; indeed, such inferences should not, in our opinion, be offered or accepted in court except in the rarest instances. Distortion may

Fig. 212.

Fig. 213.

Figs. 212 and 213.—Fracture of Ankle, showing the Neccessity of Obtaining at least Two Views. Fig. 212, which represents a lateral view of the ankle, shows practically no deformity; while the antero-posterior view (Fig. 213) reveals much displacement. (Original.)

immensely exaggerate the deformity, and nature often restores perfect function to imperfectly apposed fragments.

Tuberculosis.

This represents, perhaps, the most common bone disease. It is thought by many of our best pathologists to be always primary in the bone. If this is so, the x-rays repeatedly fail to demonstrate these early foci. It is not *unusual* in the radiograph to find a distinct focus in the bone, but it is *more usual* to discover first the thickened capsule, later the erosion of the articular surfaces, and finally the real destruction of bone.

The focus of disease, when found, usually gives little evidence, from the x-ray point of view, of any inflammatory bone process about it, and appears often as a thin-walled cavity containing more or less calcified matter (Fig. 214).

There are other strong evidences of tuberculosis where these more striking conditions are absent. Even before we can demonstrate the atrophy of the soft parts, which is so constant an accompaniment clinically, we are able, in

FIG. 214.—Tuberculosis of last Sacral Vertebra. Irregular thin-walled cavity seen with bone destruction and involvement of lumbo-sacral articulation. (Original.)

the negative, to discover atrophy in the bone in the very early stages of the disease, often long before we find any distinct focus. This is of the greatest importance in the cases of early hip disease; and here again we must have the unaffected side for comparison, preferably taken on the same plate and always with the Crookes tube focussed over the median line of the body.

The lessened shadow cast by the bone of the affected side, owing to the diminution in the lime salts and the slightly smaller diameter of the shaft, should make one very suspicious of tuberculous disease (Fig. 215).

Tuberculosis in a joint before the destructive stage ensues represents a marked irritation and, of course, often stimulates epiphyseal growth. Here we find the explanation of the often-noted fact that in the early stages of tumor albus and hip disease the affected limb is actually longer than that of the sound

Fig. 215.—Tuberculosis of Hip on the Left Side. Involvement of acetabulum and head of femur. Atrophy, especially of neck, but also of shaft of bone. (Original.)

side. In the negative we find, as an aid in reaching a correct interpretation, the characteristic enlargement and squaring of the epiphyses (Figs. 216 and 217).

As the process advances destruction is the most characteristic feature. The bone breaks down, small sequestra are formed, dislocations occur, and detritus is thrown out, with or without circumscribed abscess formation (see Fig. 218).

Finally comes repair, with new bone formation and ankylosis, or else a new joint with more or less motion and sometimes good functional adaptability (Fig. 219).

Fig. 216.—Early Tuberculosis of Knee on the Right Side. No bony focus seen. Marked thickening of capsule. Atrophy of structure of shafts. Characteristic enlargement and squaring of epiphyses. Compare with Fig. 217. (Original.)

Fig. 217.—Quiescent Tuberculosis of Knee. Same case as that shown in Fig. 216, after treatment. Acute swelling has subsided. Some atrophy of size and structure of shafts on the right side. Epiphyses still squared and enlarged. (Original.)

In the radiographs, especially those of the smaller joints, the thinning of the cortex of the neighboring bones gives them the appearance as if their outlines had been pencilled and a fine line drawn about them. Though this occurs in other conditions of atrophy—as, for example, in anterior poliomyelitis—

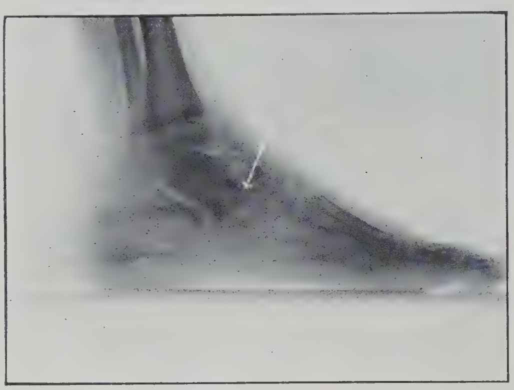


FIG. 218.—Tuberculosis of Tarsus. Destruction of scaphoid. Involvement of astragalus and cuboid. Marked structural bone atrophy. (Original.)

FIG. 219.—End Result of Tuberculosis of Hip on the Right Side. Atrophy of size and structure persist. New joint cavity and weight-bearing pillar formed. Over one-half normal joint motions. (Original.)

it is met so constantly in tuberculosis, as a result of the atrophy due to the disease, that it is almost characteristic. A form of tuberculosis called caries sicca has been described. The x-ray evidences of this condition are deep grooves in the bone at the attachment of the capsule about the anatomical neck. These

grooves are formed from an ingrowth of tough, dry granulation tissue. The joint becomes gradually obliterated. We possess no illustrative plates.

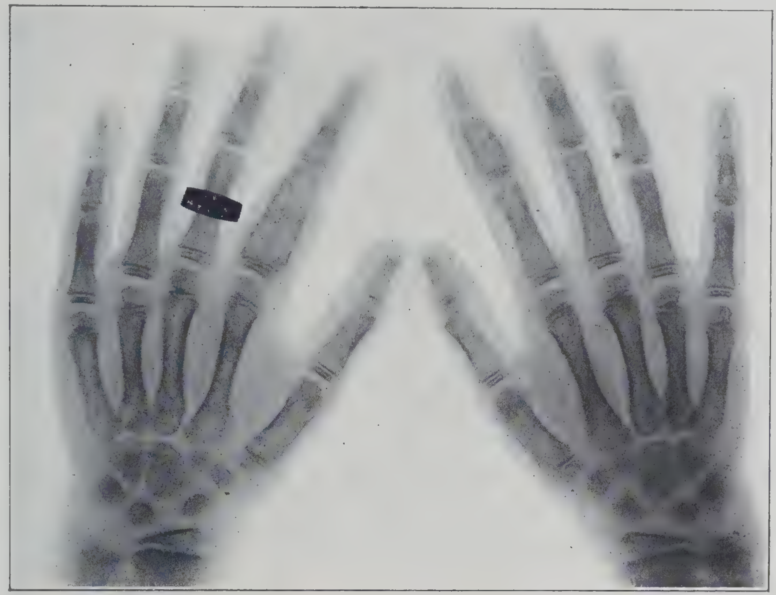


FIG. 220.—Tuberculous Dactylitis. Spinæ ventosæ. The pharanges of both index fingers—the proximal on the left, the middle on the right—show cyst-like formations with irregular and few trabeculæ. (Original.)

The spinæ ventosæ are well illustrated by Fig. 220, which was taken from a case of a tuberculous nature.

OSTEOMYELITIS.

In contrasting osteomyelitis with tuberculosis it may be said that while tuberculosis most commonly affects articular surfaces, osteomyelitis rarely does so. It is often found in close proximity to joints, but, as a rule, there is no involvement of the articular surfaces.

Except in the very long standing cases in which actual disuse has a chance to play a part, we rarely find in osteomyelitis the bone atrophy which is so constant an accompaniment of tuberculosis (Figs. 221 and 222).

There are to be seen, especially in the more chronic cases, an actual thickening of the bone cortex and a ring of bone about the osteomyelitic cavity, which is more resistant to the passage of the x-rays than the rest of the shaft.

The sequestra found are often large and of considerable x-ray density.

The two processes resemble each other in that they are both destructive at some stage, and also in the fact that at times they exhibit the characteristics of an almost malignant process, showing a tendency to fresh outbreaks after *very long* periods of apparent cure.

In considering the x-ray evidences of osteomyelitis, it is perhaps better to describe types rather than stages, for osteomyelitis is a disease of so greatly varying an etiology that it possesses no typical stages.

(1) There is a type of acute circumscribed osteomyelitis which it is often difficult to distinguish clinically from tuberculosis. The solution of the difficulty constitutes one of the most satisfactory x-ray diagnoses, for in this case

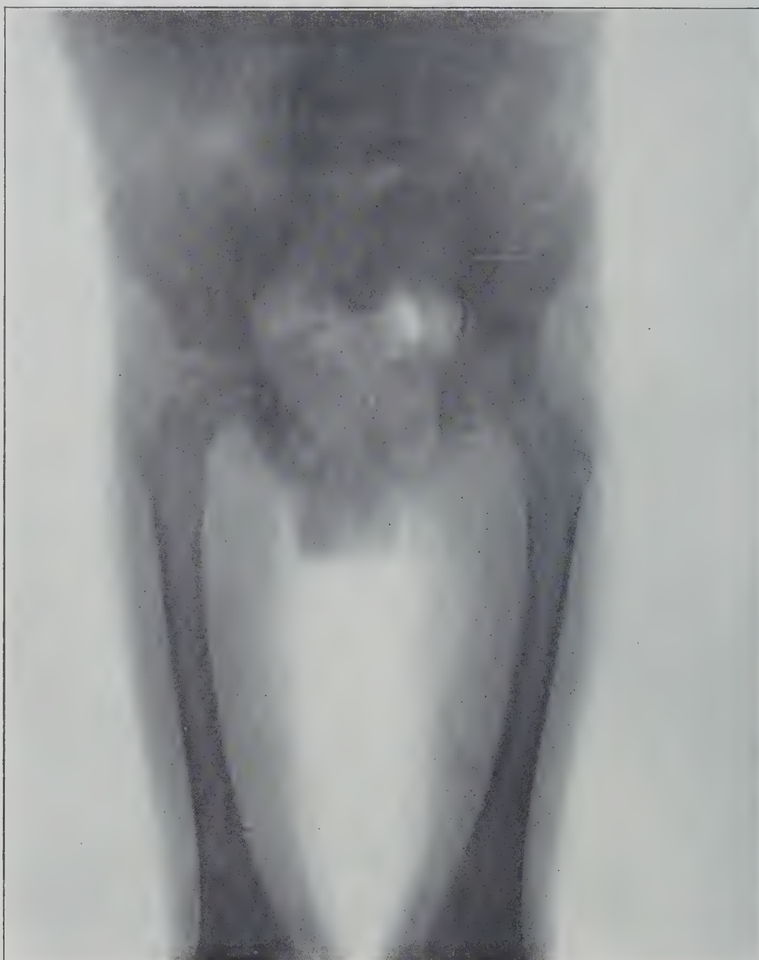


Fig. 221.—Osteomyelitis. Femoral neck on the right side snows new bone deposit along outer side. No involvement of articular surface. No atrophy of shaft. (Original.)

the dictum of the method is almost absolute and the simplification of treatment in the way of operative measures is very great. The disease is comparatively common.

In all the cases of this nature that we have seen, a pure culture of either the Staphylococcus pyogenes aureus or the Staphylococcus pyogenes albus has been obtained. The lesions which they cause are the small localized cavities

or bone furuncles. They are often chronic in character, and at times are accompanied by little external evidence of the bone process. In the radiograph the small single or multiple cavities are, as a rule, easily seen, but in certain positions they are obscured by an overlying cortex. They occur commonly near the joint

FIG. 222.—End Result of Osteomyelitis of Hip on the Left Side. No atrophy of size or structure. No involvement of articular surfaces. New bony overgrowth as well as partial destruction of great trochanter. Compare with Fig. 219. (Original.)

FIG. 223.—Circumscribed Osteomyelitic Bone Cavity in Lower End of Tibia and at Epiphyseal Line. "Bone Furuncle." Culture of Staphylococcus pyogenes aureus. Compare with Fig. 224. (Original.)

or even at the epiphyseal line, and they have thickened walls of distinct outline (Figs. 223 and 224).

(2) A far more serious condition is the acute type, which is much more

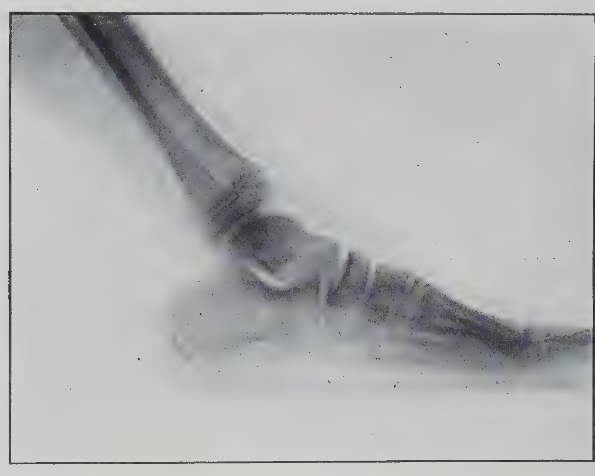


Fig. 224.—Post-operative Result One Year Later, in case shown in Fig. 223. Cavity space filled with new trabeculæ. (Original.)

Fig. 225.

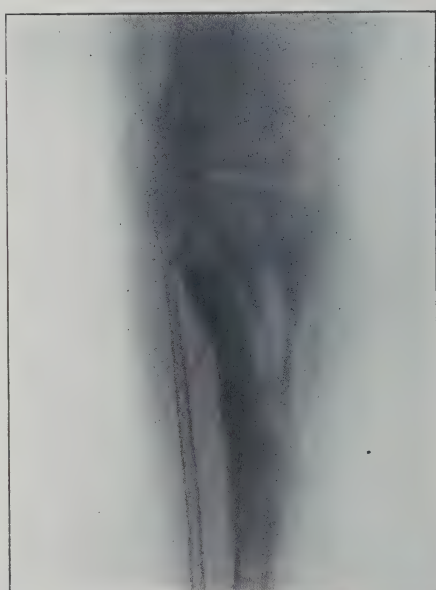


Fig. 226.

Fig. 225.—Diffuse Osteomyelitis Following Measles. Great destruction of bone, the entire shaft appearing sequestrated. Much new bony overgrowth. (Original.)

Fig. 226.—Diffuse Osteomyelitis Undergoing Healing Process. Large sequestrum forming. Same case as Fig. 225 and Fig. 227. (Original.)

diffuse, often involving nearly the whole shaft of a bone, and is at once evident in the radiograph (Figs. 225, 226, and 227). This is the type to which the term acute infectious osteomyelitis is usually applied.

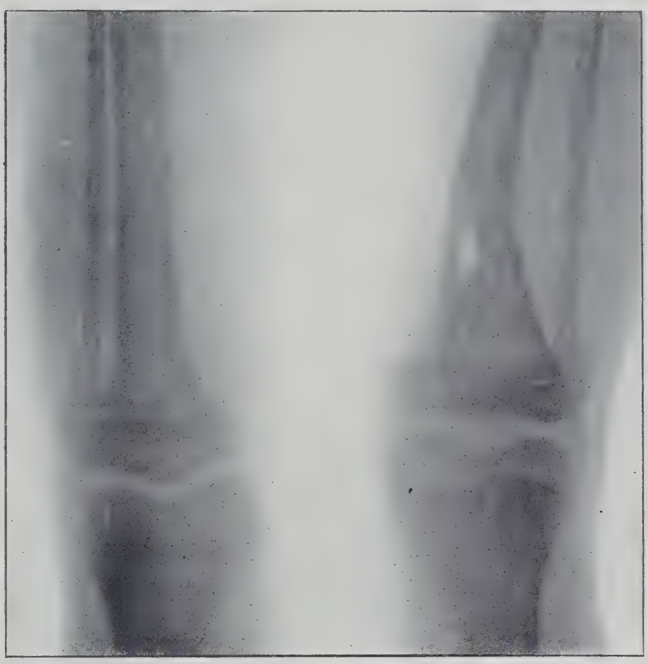


FIG. 227.—End Result of Osteomyelitis following Measles. Same case as Fig. 225 and Fig. 226. (Original.)

FIG. 228.—Chronic Osteomyelitis of Upper Portion of Tibia. Faint outline of cavity containing sequestrum seen. Great cortical thickening about cavity. (Original.)

(3) There are some writers who claim that there is a chronic circumscribed type of osteomyelitis—a form which is at times most obscure. In such cases the x-ray should help greatly in the diagnosis. The disease is to be distinguished from the first type only by the fact that the cavities are of large size, that they often lie in very dense bone, and that they are to be seen only in plates of great clearness and which show much bone detail (see Fig. 228). It will be noticed that, in the case here illustrated, the articular surfaces are free, but that the proximity of the joint has allowed the x-ray to reveal the true nature of a condition which had been treated for twenty years as rheumatic pain.

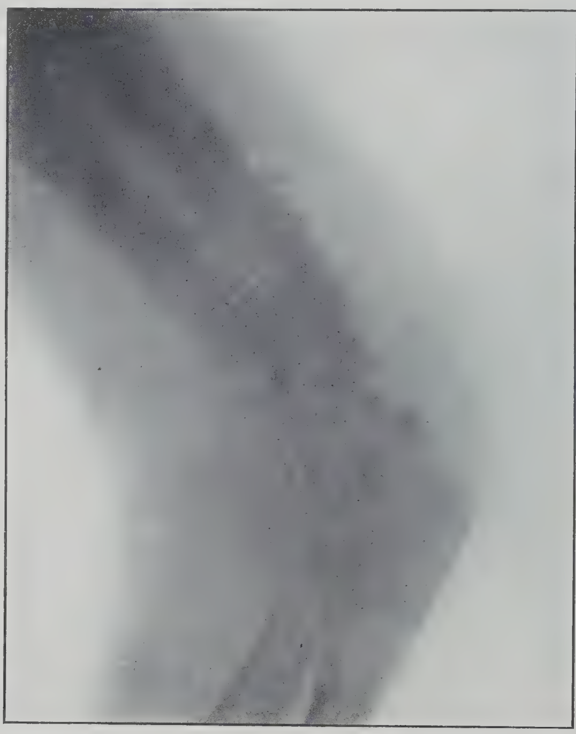


Fig. 229.—Diffuse Chronic Osteomyelitis of Humerus. Involvement of joint. Resemblance to a malignant or specific process. Diagnosis confirmed by operation. (Original.)

(4) Chronic diffuse osteomyelitis is a type more rarely encountered. Clinically the diagnosis is often difficult to establish; and, so far as this may be accomplished by the aid of the x-ray, it is sometimes impossible to distinguish the disease from one of a malignant nature (Fig. 229). There may, in these cases, be so much cortical thickening that all evidences of cavity formation are completely obscured. It is here that, with the radiograph alone, the differentiation from a specific lesion may not be practicable.

Chronic Non-Tuberculous Arthritis.

There has been much confusion, among writers on the subject of diseases of the joints, as to what terms should be applied to the different forms of

Fig. 230.—Atrophic Arthritis. General bone atrophy. Localized erosions and loss of substance can be seen most clearly in the carpal and radio-carpal articulations. Subluxations of phalanges. (Original.)

Fig. 231.—Hypertrophic Arthritis. Overgrowths of bone seen on anterior aspect of head of tibia and superior border of patella.

chronic rheumatic disease. Thus, for example, some authorities class them together under the single head of arthritis deformans, maintaining that they represent different stages of one and the same disease. In America, on the

FIG. 232.—Atrophic Arthritis of Knee. Weak bone shadow as compared to soft parts. Normal joint space absent because of loss of cartilage substance. Erosions on under surface of patella. (Original.)

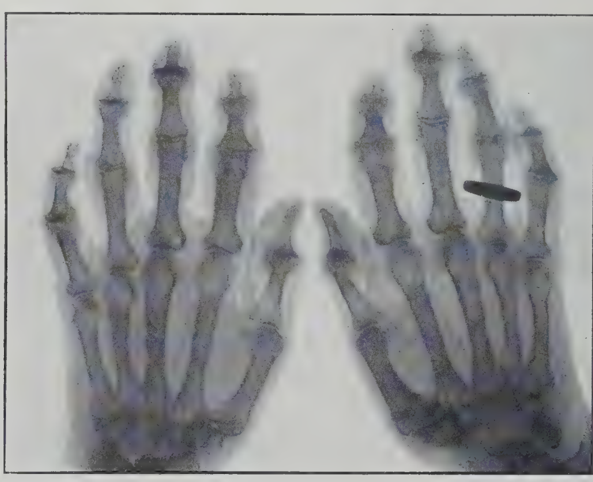


FIG. 233.—Hypertrophic Arthritis. Strong bone shadow. Marked overgrowths can be most clearly seen on terminal and some mid-phalangeal articulations. Some joint surfaces destroyed by overgrowth of bone and cartilage. (Original.)

other hand, there are many who regard them as more or less separate and independent diseases. So far as it is possible to judge from examinations made with the aid of the x-ray, the statement is warranted that the so-called

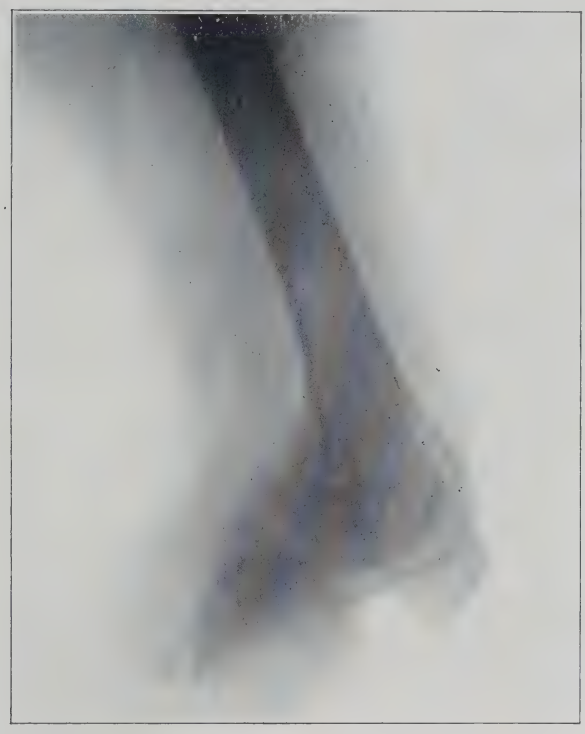


FIG. 234.—Hypertrophic Arthritis. Well-marked overgrowths seen on femoral condyles and on the under surface of patella. Thickened cartilage in popliteal space. (Original.)

FIG. 235.—Infectious Arthritis. Marked periarticular swelling. General diffuse atrophy, but no erosions or definite impairment of joint surfaces. No hypertrophy. (Original.)

atrophic or rheumatoid arthritis and the hypertrophic form of the disease, or osteo-arthritis, almost always, even in the early stages, manifest distinct

Fig. 236.—Infectious Arthritis. Same case as that shown in Fig. 235, but one year later. Periarticular swelling has largely disappeared. No essential involvement of joint surfaces. No hypertrophy. Some general atrophy from lack of use. (Original.)

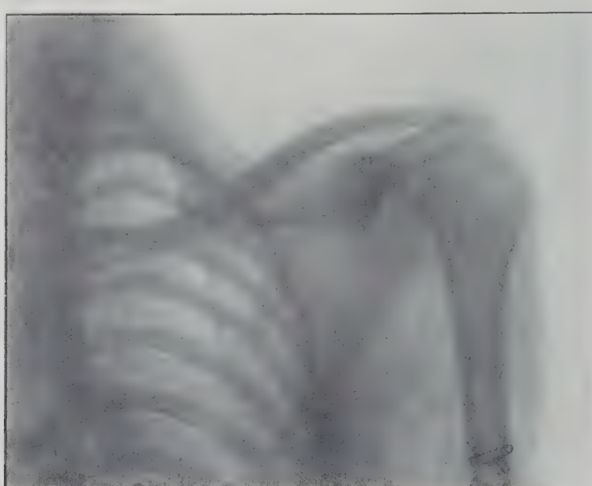


Fig. 237.—Infectious Arthritis. Complete fibrous joint ankylosis; neither atrophy nor hypertrophy. No change in articular surfaces. (Original.)

and separate conditions. One represents atrophy, the other hypertrophy; one a destruction of cartilage and a loss of substance, the other thickening of

cartilage, the deposition of lime salts, and actual outgrowth of new bone. Watched from the onset of the first symptoms the two diseases seem to differ essentially. One is occasionally superimposed upon the other just as scarlet fever may be accompanied by diphtheria. In these rather unusual cases the patients themselves will, as a rule, recognize them as distinct processes. The interpretation of the negatives, in suspected or pronounced cases of these diseases, is interesting and not difficult (see Figs. 230–234).

Fig. 238.—True Gout. Definite loss of substance of shafts of proximal phalanges of index and little fingers. Several joints involved. No tophi distinguishable. (Original.)

Infectious Arthritis.

A large number of joint lesions, single and multiple, are unquestionably produced by some toxin or are due to a true bacterial infection. To these, Goldthwait has given the name of "infectious arthritis." It is possible to distinguish these by the x-rays, as well as clinically. Indeed, when the clinical diagnosis is in doubt, the radiograph often furnishes conclusive evidence. The joint lesion in the active stage represents neither essential atrophy nor hypertrophy of bone structures. The capsule is thickened and infiltrated,

with or without excess of fluid in the joint, but with no erosion of joint surfaces (see Figs. 235–237).

The x-rays would suggest that the so-called Still's disease represents an identical or certainly analogous process.

Fig. 239.—Hereditary Syphilitic Disease. Late manifestations. Juxta-epiphyseal form. Epiphyses little affected. Confluent areas of porosity in diaphyses of both tibiæ. Increase in cortical bone, and areas of bone deposit beneath the periosteum in diaphysis of femur on the right side. (Original.)

Gout.

True gout seems to be in a class by itself. The tophi are scarcely distinguishable in the negative, but the loss of substance of the shafts of the bones, as well as the involvement of the joint surfaces, is, in the advanced stages, characteristic (Fig. 238).

In interpreting the plates of any of these chronic articular diseases, the stage of the process must be considered if confusion is to be avoided. Thus,

FIG. 240.—Hereditary Syphilitic Disease. Diffuse cortical thickening of mid-tibial and lower fibular shaft. New bone deposit beneath the periosteum giving rise to sabre-shaped bone. Tendency along tibial crest to the formation of so-called "bone-blisters." (Original.)

FIG. 241.—Periostitis Albumosa. Rough irregular bone deposit about the end of the radius, with areas of rarefaction in the diaphysis. Elbow and ankle of same case showed similar changes. (Original.)

an atrophic process, which has become quiescent in any joint, may make feeble attempts at repair and actually throw out new bone at the points of primary erosion; *vice versa*, a hypertrophic process, carried to the point of ankylosing a joint, may bring such pressure to bear on cartilaginous surfaces that erosions

Fig. 242.—Hereditary Syphilitic Disease. Probable bone gumma in late form of the disease. Increase in cortical bone and lighter shadow of newly formed calcareous deposit on the left side. Circumscribed lesion. (Original.)

occur, and the bones, from mere disuse, show atrophy. The same holds true of the infectious types.

Despite these facts, in the majority of cases the *x*-ray negative is of almost conclusive value in differentiating the types and revealing the essential nature of the process.

Syphilitic Disease.

The bone lesions which occur as the result of luetic infection are numerous and varied. We shall attempt to describe the x-ray appearances of only the common types.

We have personally seen few pathological appearances in the x-ray plates of bones taken in the secondary stage of the disease. The hereditary and con-

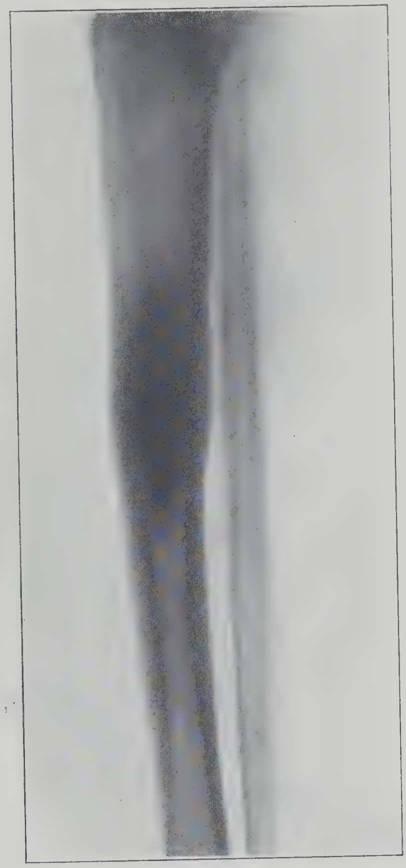


Fig. 243.—Tertiary Syphilitic Disease. Circumscribed bone gumma. Marked increase in density of cortex; apparent invasion of the medulla. (Original.)

genital forms and the tertiary lesions give most striking pictures and offer a large opportunity for diagnosis.

The hereditary forms are divided into the early and the late, the former appearing soon after birth and resembling clinically rickets. Pathologically, the condition is represented by the presence of gelatinous masses beneath the peri-

Fig. 244.—Hereditary Syphilitic Disease. Typical diffuse syphilitic osteomyelitis, showing in different regions a deposit of bone beneath the periosteum, thickening of cortical bone, rough new overgrowth, and bone necrosis. (Original.)

Fig. 245.—Hereditary Syphilitic Disease. Same case as that shown in Fig. 244, after anti-syphilitic treatment covering a period of two years. (Original.)

osteum and at the epiphyseal line, with sometimes true fractures or separations of the epiphyses. Among the other alterations there is said to be a thickening of the cortex and periosteum with gelatinous deposit between the two. We personally have never seen a good x-ray plate of this condition.

One of the later hereditary forms of syphilis—the juxta-epiphyseal lesions—deserves a special mention. In these lesions areas of bone necrosis accompany a deposit of bone beneath the periosteum and some thickening of the cortex.

Fig. 246.—Hereditary Syphilitic Disease. New deposit of bone beneath the periosteum of one of the metacarpal bones. Old line of cortex seen. (Original.)

Were it not for this overgrowth and for the fact that the articular surfaces are free, the condition might be confused with a diffuse tuberculosis. (See Fig. 239)

Thickening of the cortex and the deposit of bone beneath the periosteum are the most characteristic and common evidences in the late hereditary and in the tertiary forms of the disease. They give rise, for example, to the sabre-shaped tibiæ seen clinically (Fig. 240).

It is by no means rare to find along the shaft of this thickened bone small areas of rarefaction with a cap of dense bone rising up over them. They have been well named by Codman "bone blisters."

Fig. 247.—Charcot's or Tabetic Joint. Antero-posterior view. Marked loss of substance of inner femoral condyle and tibial head on right side. Compare more or less atrophied bone with normal unaffected knee on the left side. (Original.)

Fig. 248.—Charcot's or Tabetic Joint. Semilateral view. Bone destruction with loose masses containing calcareous matter. Irregular articular surface of tibia. (Original.)

The bone deposit beneath the periosteum, spoken of often as periostitis, appears as a faint localized bulging along the shaft, not unlike early callus.

The only conditions likely to be confused with these lesions are those which are observed in the early stages of an osteomyelitis proper or in that form of the disease which is known as periostitis albumosa (Fig. 241). The latter condition shows itself as an irregular deposit of calcareous matter outside the cortex in proximity to the joint. In the single case that has come under our observation several bones were affected. The normal bone ridges at the points of insertion of muscles or where ligamentous structures are attached must not be mistaken for this calcareous deposit.

Fig. 249.—Exostosis Causing Fracture of Fibula. Osteoma medullosum. (Original.)

The occasional periostitis that occurs after typhoid may resemble closely the so-called bone blisters, though without the accompanying cortical thickening. This cortical thickening is usually more marked along one side of the bone, often encroaching on the medullary cavity. The differentiation from an old osteomyelitis, by means of the negative alone, may be difficult, and not a few cases of Paget's disease, or of osteitis deformans, have been diagnosed as of a syphilitic nature from the presence of this cortical thickening. Paget's disease is

rarely confined to one bone except in the earliest stages; syphilitic disease often is. The areas of rarefaction commonly observed in osteitis deformans are not characteristically seen in this type of specific lesion.

The bone gummata observed in the tertiary stage of syphilis are both superficial and deep, the former occurring between the bone and the periosteum and causing ulceration of the cortex. These gummata may occur side by side, or they may merge the one into the other through radiating connecting bands. The

Fig. 250.—Exostosis of Femoral Shaft. Osteoma spongiosum. Structure of soft parts well shown. (Original.)

deep gummata may occur in any part of the bone and may lead to fracture. The new bone thrown out on the side of the cortex next the lesion may be of ivory-like hardness, casting a very dense shadow (Figs. 242 and 243).

In **diffuse syphilitic osteomyelitis** the bone may be doubled or tripled in volume, with numerous osteophytes (Fig. 244 and 245).

Charcot's or tabetic joints should probably be looked upon as true arthropathies of perhaps neuropathic origin, rather than as evidences of active specific

Fig. 251.—Enchondromata. Index finger of left hand shows most marked overgrowths. Small buds seen on the proximal phalanges, on the left middle finger, and on the thumb. (Original.)

Fig. 252.—Medullary Sarcoma of the Tibia, of the Myelogenous or Giant-celled Variety. Marked cystic formation in the upper part of the tibia. (Original.)

infection. They are most characteristic in the x-ray as well as in their clinical behavior. The instability of the part and the frequent tremor which sets in when an attempt at fixation is made, render it difficult to obtain clear radiographs. When they have been successfully taken they show great disorganization of the joint, with more or less destruction of large portions of the articular and juxta-articular bone, and the presence, in the joint, of apparently loose masses of detritus often containing lime salts.

FIG. 253.—Osteo-sarcoma and Osteitis Deformans. Periosteal sarcoma developing in a very advanced case of osteitis deformans (well seen in tibia). Sarcoma involves lower end of femur and popliteal space. (Original.)

The boggy-feeling joint often gives a foggy-looking radiograph (Figs. 247 and 248).

Syphilitic dactylitis perhaps deserves a separate heading. So far as our observation goes, it is not a difficult matter to confuse the disease with tuberculosis. The distinguishing features are: in syphilis there is less atrophy of structure and the articular surfaces are less often involved. If

loss of substance occurs, the bone ulcer will present clear edges and a punched-out appearance (Fig. 246). A tuberculous dactylitis is by far the commoner form.

Exostoses or Osteomata, and Enchondromata.

These conditions need little experience for their interpretation, since skiagraphs taken in different planes will usually definitely locate and determine the size of these bland overgrowths.

Fig. 254.—Secondary Epithelioma of the Tibia and Tarsus. Cancerous osteomalacia, showing great destruction and atrophy. (Original.)

They are by no means always of the same structure, which has given rise to the self-explanatory terms "osteoma eburneum," "osteoma spongiosum," and "osteoma medullosum" (Figs. 249 and 250).

Fig. 255.—Osteitis Deformans or Paget's Disease. Characteristic changes seen in the bones of both forearms and in the third and fourth metacarpals of both hands. Bowing of bones, regions of greatly thickened cortex, and areas of rarefaction. (Original.)

The enchondromata, or chondral osteomata, appear in the neighborhood of the joints as irregular masses of cartilage containing calcareous matter, or as true exostoses. They are supposedly formed from bits of cartilage left behind in irregular epiphyseal development. They are usually multiple and are found in various joint regions (Fig. 251).

Bone Tumors.

Sarcomata are by far the commonest bone tumors, and the distinguishing characteristic of many of the types can be discovered by the *x*-rays.

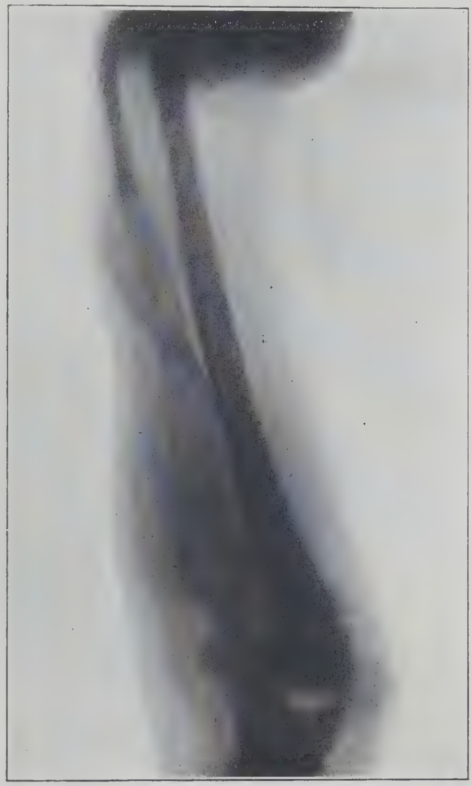


Fig. 256.—Osteomalacia. Marked cystic formation in radius with great loss of lime salts. Acute bend of both radius and ulna at the wrist. (Original.)

The myelogenous or medullary giant-celled sarcoma is prone to develop cysts in bone, and these cysts are often filled with blood and comprise the so-called bone aneurisms (Fig. 252).

The tumors of the osteoid or periosteal spindle-celled type present themselves in the form of smooth, often dense swellings that spring from the superficial surface of the bone and are intimately connected with it, although not infrequently the old cortical outline can still be made out. Their radiating appearance is often recognizable (Fig. 253).

The multiple myelomata, usually round-celled, tend to soften and disintegrate the bone, which is then apt to undergo pathologic fracture.

We are unfamiliar with the appearances of the angio-sarcomata.

Epithelial neoplasms are probably rarely if ever primary in bone, but the secondary forms and metastatic growths cause most extensive bone destruction and are well described as "cancerous osteomalacia" (Fig. 254).

Osteitis Deformans or Paget's Disease.

Once familiar with the typical gross appearances of osteitis deformans, one can hardly mistake a well-marked case for any other condition. The x-ray

Fig. 257.—Rachitis. Wide cloudy space between the diaphysis and the epiphysis. Small size of epiphyseal bone centre. Typical enlargement of contour seen at the epiphyseal line. (Original.)

plate is equally characteristic and often furnishes the most definite and conclusive evidence in an unsuspected case. It is not difficult to mistake the indefinite symptoms of bone pain and slight thickening of an early case for specific disease or osteomyelitis. If x-ray evidence is sought, it will usually be found that other bones of the body show typical lesions often quite unsuspected

FIG. 258.—Severe Rachitis. Slight bowing of femur on the left side. Markedly irregular epiphyseal line. Gradual flaring of diaphyses. Extreme coxa vara. (Original.)

by the patient. Periosteal and cortical thickening go hand-in-hand with circumscribed and irregular areas of rarefaction. The bowing of the bones always sooner or later appears, and the joint surfaces, while often showing hypertrophic nodes or spurs, are not otherwise affected. The frequently concomitant sign of arteriosclerosis reveals, in many cases, the calcified walls of the larger vessels in exquisite outline. (See Fig. 255.)

Osteomalacia.

Cystic formations with medullary and cortical destruction, acute bowings and angular bendings of bones in osteomalacia give us a picture which can

Fig. 259.—Chondrodystrophia. Adult case. Wide sudden expanding of ends of bones. Short stubby shafts. No bowing in the bones affected. (Original.)

hardly be confused with other conditions in a well-marked case. The large amount of partly calcified callus and cartilage at the seat of spontaneous fracture is also characteristic, and so too is the marked diminution of lime salts in the bones affected (Fig. 256).

Rachitis and Chondrodystrophia Fœtalis.

The only condition likely to be confused with a typical rachitis is the misnamed fœtal rickets, or the condition more properly designated chondrodystrophia fœtalis.

In both diseases the diaphyseal ends are flaring; in rachitis this enlargement shows an easy curve, while in chondrodystrophia the expansion is of an abrupt character.

In severe rachitis there exists a wide cloudy space between the epiphysis and the diaphysis, and the adjoining surfaces are irregular. The contour of the epiphysis toward the joint is smooth, but the bony centre is usually smaller than normal. The shafts are of normal length, though apparently shortened from the characteristic bowing. The cortical bone will be found dense on the concavity of the curve, and sometimes indefinitely porous on the convex (Figs. 257 and 258).

Fig. 260.—Adolescent Rickets. Coxa vara. Well-nourished boy of fifteen years. Wide, irregular epiphyseal line. Axis of neck of femur is much more horizontal to the axis of the shaft than is normal. (Original.)

In chondrodystrophia there exists a rather narrow but fairly regular line between the epiphysis and the diaphysis. The shafts are short, unbowed, and usually thick, and the resultant clinical dwarfing is thus explained (Fig. 259).

Adolescent Rickets. Coxa Vara.

Whether the above condition is really a late manifestation of a rachitic process or a separate disease, the x-ray findings are conclusive as to the bent

FIG. 261.—Osteogenesis Imperfecta. Periosteal dysplasia. Almost entire absence of cortical bone. Numerous fractures. (Original.)

FIG. 262.—Acromegaly. Comparatively slight bony enlargement. Wide joint spaces. A few small osteophytes on metacarpal heads. Great enlargement of soft parts. Compare with Fig. 263. (Original.)

FIG. 263.—Giantism. General enlargement, but no other peculiar characteristics. Compare with Fig. 262. (Original.)

FIG. 264.—Congenital Dislocation of the Hip on the Left Side. Rudimentary acetabulum. Small epiphysis of the head of the femur; atrophy of the shaft. (Original.)

FIG. 265.—Congenital Malformation of the Vertebræ. Two wedge-shaped and several malformed vertebræ. Marked lateral curvature (torticollis). Case was recommended for operation, the futility of which is shown by the *x*-ray. (Original.)

FIG. 266.—Congenital Elevation of the Scapulæ. Showing curvature of the spine, wedge-shaped vertebræ, cervical rib, and malformed scapulæ, both elevated. (Original.)

FIG. 267.—True Bony Ankylosis of Knee Joint. Continuous bone trabeculæ to bridge old joint space. (Original.)

FIG. 268.—Raynaud's Disease. Absorption of different amounts of the terminal phalanges without other evident change in bone structure. (Original.)

FIG. 269.—Calcareous Deposit in the Course of an Acute Infectious Process. Deposition of what seem to be lime salts above the humeral head. Joint lines clear. Subsequent x-ray picture, taken two months later, shows entire disappearance of calcareous matter. (Original.)

Fig. 270.—Comparative Bone Atrophy. Bones in hand on the right side cast less dense shadow, and structure shows thinning of trabeculæ. Disuse from a neuritis. (Original.)

neck and the frequent appearance of a solution of continuity between the epiphysis and the shaft resembling an intracapsular fracture. The bone appears of nearly normal structure, with perhaps a slight diminution in the amount of lime salts (Fig. 260).

Fragilitas Ossium or Periosteal Dysplasia, and Osteogenesis Imperfecta.

These two conditions are both characterized by insufficiency of cortical bone and numerous fractures.

Fragilitas ossium occurs after birth and is to be distinguished from other causes of brittle bones.

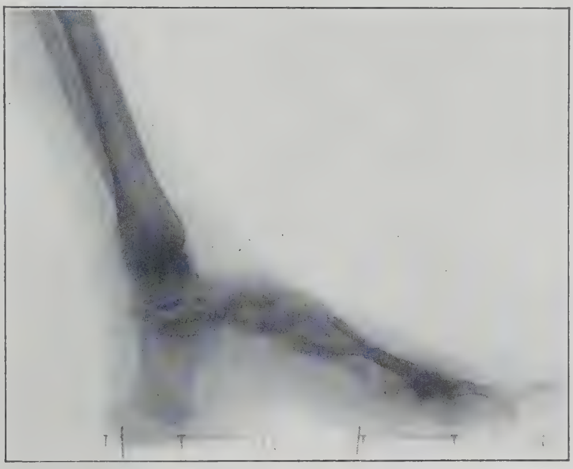


Fig. 271.—Bone Atrophy. Pes calcaneus following anterior poliomyelitis. Considerable general atrophy of size and some of structure. Leg imperfectly used. (Original.)

Osteogenesis imperfecta is congenital, the epiphyses appearing nearly normal while the shafts manifest periosteal dysplasia and an almost entire absence of cortical bone, with frequent fractures or bendings (Fig. 261).

Acromegaly.

In this disease there are enlargements of both the soft parts and the bony structures; the enlargement of the latter, however, being only slight in degree. In addition, osteophytes may appear in the neighborhood of joints, but the articular surfaces are, as a rule, free. A striking feature is the wide joint spaces. (See Fig. 262, and compare with Fig. 263, the latter being a case of giantism.)

Ostéoarthropathie Hypertrophiante Pneumique.

This rare condition may be confused with both syphilitic disease and osteitis deformans. It is said that there may be seen an outside less dense layer

of newly formed periosteal bone all along the shaft. This layer of bone, which has a striated appearance, does not completely obscure the old cortical line.

The only radiograph at our disposal shows this very imperfectly.

Elephantiasis Ossium.

This name has been applied to an enlargement of bones produced by periosteal irritation. It occurs most markedly in cases in which there has been an antecedent sepsis.

Fig. 272.—Bone Atrophy. Anterior poliomyelitis. Bones on the left side show considerable atrophy of size, but little of structure. Leg in constant use. (Original.)

Congenital Malformations.

X-ray plates may satisfactorily determine the bony depth of the acetabular cavity and the contour of the head of the femur in cases of congenital dislocation of the hip, and they readily differentiate this condition from an early coxa vara, with which it is easily confused (Fig. 264).

Obscure cases of torticollis will not rarely be found to have malformed, wedge-shaped vertebræ as their etiological basis, frequently associated with one or more cervical ribs (Fig. 265).

Congenital elevation of the scapula may also be clearly demonstrated by the x-ray plate (Fig. 266). The recent investigations made by Dr. Max Boehm seem to show that many cases of scoliosis have congenital abnormalities of the vertebræ.

Ankylosis.

The diagnosis of bony ankylosis should be made from the x-ray plate with very great caution, and in our opinion only when clear continuous bone trabeculæ can be seen bridging the old joint space (Fig. 267). Overlapping bone edges and dense fibrous adhesions may often otherwise deceive.

Raynaud's Disease.

The absorption of the terminal phalanges without some evidence of a repair process is characteristic. The remaining bone seems little altered in structure (Fig. 268).

Deposits of Calcareous Matter.

It is desirable to mention here a condition or a group of conditions which show in the negative a definite shadow nearly as dense as that due to bone. This shadow usually appears in close proximity to the joints and suggests an excessive hypertrophic process. The mass which may be seen in Fig. 269 developed in the course of an infectious arthritis of the shoulder, and, under antirheumatic treatment, it was found—two months later, when a second x-ray picture was taken—to have disappeared entirely. We have noted almost the same appearances in an x-ray picture of an old wen, the residue of whose contents was almost pure calcium. Doubtless other conditions will be found to give similar shadows.

Simple Bone Atrophy.

A few words should be said under this heading, although many of the conditions in which bone atrophy is an essential feature have already been described.

The condition is easily demonstrated by the x-ray if we have a normal standard of comparison, the two pictures being taken preferably at the same time and on the same plate, but in any case under the same conditions of exposure and development.

In tuberculosis it is a phenomenon of almost constant occurrence. In atrophic arthritis we always expect to find it. In certain neurotrophic disturb-

ances and in the reflex joint atrophies it is present. Senile changes in bone are shown by a general thinning of the cortex and by a diminution in the number of the trabeculæ. Simple disuse apparently always decreases the quantity of lime salts, and thus the more radiable bone casts a fainter shadow on the plate (Fig. 270). Cases of anterior poliomyelitis show the smaller size of the affected bones, which are usually of less density than the normal (Figs. 271 and 272).

We should be careful to make a distinction between atrophy of structure and atrophy of size. Both are often found together, but not rarely one is present and the other absent.

PART IV.

GENERAL SURGICAL TREATMENT.

GENERAL PRINCIPLES OF SURGICAL TREATMENT AND THE VARIOUS PROCEDURES, INSTRUMENTS, ETC., THAT FACILITATE THE APPLICATION OF THESE PRINCIPLES.

By JAMES E. MOORE, M.D., Minneapolis, Minnesota.

In 1579 Ambroise Paré, the greatest surgeon of his day, published a voluminous work on chirurgery, in which he said: "For God is my witness, and all good men know, that I have laboured fifty years with all care and pains, in the illustration and amplification of chirurgery; and that I have so certainly touched the work whereat I aimed, that antiquity may seem to have nothing wherein it may exceed us beside the glory of invention, nor posterity anything left but a certain small hope to add some things." The progress of surgery was so slow in those days that this work was the standard for over one hundred years. Seventy years after its publication in Paris it was translated into English and published in London. This same complacent spirit seems to have pervaded the profession, and progress was correspondingly slow up to within the last thirty years; since which time surgery has made more progress than in all time before, and surgeons begin to feel that the limitations of surgery are almost boundless.

Modern surgery had its beginning in 1865, when Joseph Lister, of Glasgow, Scotland, published the results of his experiments and established the "Germ Theory of Disease." In 1858 Pasteur announced to the world that fermentation and putrefaction are due to the action of living micro-organisms, and are therefore living processes. Up to this time, inflammation and suppuration had been considered necessary accompaniments of a wound. Lister reasoned that if putrefaction and fermentation were due to the presence of germs and could be prevented by their exclusion, suppuration and certain diseases must be due to their presence and could be prevented by their exclusion. He demonstrated by experiment that a wound made in a clean skin by clean hands and instruments and protected by clean dressings would heal without inflammation or suppuration. The application of this germ theory has revolutionized the practice of medicine and surgery, and has added thousands of years to human life. It has made possible the prevention and suppression of many epidemics, and has changed hospitals from pest-houses to houses of refuge. The discovery of the tubercle bacillus by Koch, published in 1882, was one of the many advances

made possible by using the germ theory as a working basis. Up to that time the term scrofula was applied to swollen lymph nodes, chronically inflamed joints, and other conditions now known to be due to the presence of the tubercle bacillus, and the condition was believed to be hereditary. Lister's theory was not enthusiastically adopted at first, but its truths soon began to impress themselves upon the profession, so that by the time Koch made his discovery they were ready to go to extremes. The great discoverer himself believed for a time that he had discovered a panacea for all who were afflicted with tuberculosis, and the majority of the profession began the use of his lymph with enthusiasm, only to be disappointed. Since that time the development of surgical technique has been rapid and steady, until it has reached such a state of perfection that we are in danger of thinking, as did Ambroise Paré, that there are but few things posterity can add. Within the experience of many surgeons still in active practice, we have passed from old-time or septic surgery to aseptic or present-day surgery. The keynote to modern surgical technique is surgical cleanliness, and this is best secured by a happy combination of the principles of antisepsis and asepsis. For a number of years during the development of our present technique our medical journals teemed with articles upon this subject, but at the present time an article on technique is considered unnecessary. This is a dangerous attitude, because a belief that we have arrived at the acme of human perfection will surely prevent progress. The great benefits vouchsafed mankind through this gospel of cleanliness are not without alloy. The fact that such wonderful things can be done through a clean wound has led to the doing of many unwarrantable things. There is too much of a tendency among young members of the profession to believe that technique is all there is of surgery, and that as soon as a man can secure healing by first intention he is a surgeon. There is decidedly too great a tendency to make a diagnosis with the knife. Operations are a very necessary part of surgery, but it is more important to decide when and when not to operate. It sometimes requires more courage to decide not to operate than to operate. The surgeon should always be bold but conservative, for conservatism is the greatest attribute of a good surgeon. The conservative surgeon will operate only when he believes that an operation is the treatment most likely to save the life of his patient or to restore him to health. He is always open to conviction, but does not accept statements because they are new, nor does he discredit established facts because they are old. It is not conservatism to refuse to operate when an operation is clearly indicated. The knife is often the greatest of conservative agents.

There are cases in which an exploration is the only possible means of making an accurate diagnosis, and it is then not only justifiable but advisable; but it should be a court of last resort. The fact that it can be done by competent surgeons with very little danger gives them no license to resort to it without first having exhausted all other known means of diagnosis. Unfortunately,

there are a good many operators, some of them very skilful too, who are not surgeons. A surgeon, except in an emergency, takes ample time to study his cases before advising for or against an operation, while a mere operator is prone to operate without this preliminary. The result is that many unnecessary and unwise operations are performed, and surgery is brought into disrepute. The surgeon watches his patients carefully after the operation, because he feels that he has not performed his whole duty until the patient is fully restored to health; but one who thinks that operations are the whole of surgery too often neglects the after-treatment, turning the patient over to the nurse. Every young man should begin by studying his cases carefully and keeping a written record of them, for in this way he accomplishes the most good for his patient and he profits by his experience. When one begins his professional career in this manner, he establishes good habits and becomes a broader and better practitioner.

At the present day, when hospitals are so numerous, no man who is ambitious to do surgery should consider his education complete without a hospital training. In after-years he will realize that his hospital experience is the best part of his education. The greater his natural endowment and ambition and the better his education, the more imperative is this practical education, because nothing is more dangerous than misguided talent and energy. Another way of getting this practical experience, which is equal to, if not better than, hospital experience, is to act as assistant to some surgeon who is doing good work.

The most of surgery should be learned before technique, but without good technique one cannot be a surgeon. The personal equation makes ofttimes the whole difference between success and failure. Some men seem to be possessed of an aseptic conscience and will always do the clean thing without giving it special thought, while others need some one specially appointed to watch them. The former, should he need a chair in the midst of an operation, will intuitively reach for it with his foot, while the latter will use his hand and will then introduce this possibly septic hand into the wound. Some operators accomplish the most perfect results attainable by a very simple technique, while others, with a most elaborate technique, fail to get good results. The one understands and practises the principles of asepsis; the other does not. The one grasps the situation; the other does not.

The best operators are not always the best surgeons, but, other things being equal, a clever operator is the best surgeon. A very skilful operator may be a poor surgeon, but a good surgeon owes it to himself and his patients to cultivate manual dexterity. A good operator works rapidly without seeming to hurry. Unseemly haste and extreme slowness are both to be depreciated. Time certainly is an element of success in a surgical operation. The best operator is the one who can do the maximum amount of good, clean work in the minimum amount of time. One who is naturally endowed with mechanical dexterity has

a very great advantage as an operator, but every one who is ambitious to become a surgeon, no matter what his natural qualifications, should make a careful study of the mechanical part of his work. The swift, gliding motion of the knife that cleaves the tissues without seeming to press upon them, the dextrous tying of a surgeon's knot, the quick, certain grasping of a spurting blood-vessel, and the gentle handling of the tissues are all evidences of good operating. Ambidexterity is a natural gift of very few, but it can and should be cultivated by every operator. Every young man should begin by using whichever hand is most convenient for the work that is to be done, no matter how clumsy he may be at first, for ambidexterity can be acquired by every operator by persistent effort, and, when once acquired, it is equivalent to an extra assistant. A good surgeon is always careful not to destroy or injure any tissue unnecessarily, and at the same time he uses every means at his command to avoid or destroy bacteria. The anatomical operating methods of our forefathers are infinitely superior to the rough methods too often practised to-day. Blunt dissections by fingers or instruments should be employed only when the exigencies of the case demand it.

Modern surgical technique is made up of many little details. Each detail is a link of a chain which when completed makes a perfect surgical operation, and every necessary detail omitted makes a weak spot in the chain. The measure of perfection of an operation, however, is the simplicity of its detail. The operator who knows least of aseptic surgical technique is the one who makes the greatest display. Simplicity of technique should be cultivated, because every unnecessary detail is an element of weakness which tends to bring the method into disrepute. A novice is much more likely to succeed with a simple technique than with an elaborate one. When the method is too elaborate he is apt to forget some part or to conclude that it is unnecessary, and he may be incompetent to decide what is and what is not essential, for what is essential can be established only by bacteriologic experiments. When one without initiative, but who is simply doing what he has been told to do, discovers accidentally that he can omit some detail which he has been taught was essential, without serious consequences, he is very apt to conclude that there are others which can be omitted, and the end will be disastrous. When the minimum requirements for aseptic surgery are employed, they appeal to the beginner as rational and therefore essential, and he will not neglect them. Just what the minimum requirements are cannot be definitely settled, because they differ with men and environment. Each operator should follow a definite routine in his asepsis, and he will thus learn what his person and his environment require; and, having once established a technique which brings about the desired results, he should adhere to it until he is satisfied he can make a change for the better. Change for sake of change should be avoided. No one can be a safe surgeon who is not thoroughly imbued with the principles of asepsis, for without this he will be doing things

just because some one has told him to, and cannot appreciate their value. When a surgeon's ideas of cleanliness are the same as those of a mother when she washes her child's face, he is certain to be careless and to handle unsterilized articles after he has prepared his hands; but when he thoroughly understands that the cleansing of hands and everything connected with an operation means sterilization or the removal of bacteria, he will understand the necessity for avoiding contact with unsterilized articles, and that every unsterilized article is a possible source of infection.

Sepsis is the condition following the introduction of pyogenic bacteria into a wound. Sepsis is caused only by bacterial invasion.

Antisepsis is the term used for designating the various methods employed to destroy or inhibit the growth of bacteria in a wound.

Antiseptics are the drugs and other agents employed to destroy or inhibit bacteria.

Antiseptic surgery is the name given to the technique taught by Lister which was practised for many years. Lister believed that the air was the principal carrier of germs, and that it was necessary, in the case of every wound exposed to that element, to use antiseptics for the purpose of destroying these germs. He impregnated the atmosphere of the operating-room with carbolic spray, and it was made to play over the hands of the operator and the field of operation constantly. Instruments and ligatures were prepared by immersion in carbolic lotion. The spray was very disagreeable to the operator and sometimes produced some unpleasant effects in the patient, so that it was soon abandoned. Before the establishment of the germ theory surgeons believed that in some mysterious way the air caused inflammation in a wound. In 1841 Stromeyer performed subcutaneous tenotomy, and this was followed by many subcutaneous operations, the object being to prevent the entrance of air. It has been conclusively demonstrated that the atmosphere plays a minor part in wound infection, but a dust-laden atmosphere is to be avoided, because every particle of dust may act as a carrier of germs. This knowledge has led to a gradual change, so that the term applied to modern surgical technique is aseptic surgery.

Asepsis means the absence of living pathogenic bacteria, and when practising *aseptic surgery* we endeavor to destroy and remove all micro-organisms from everything connected with the operation, so that we may have an aseptic wound. It is claimed that an absolutely aseptic wound is an impossible condition, but the natural resisting powers of the tissues are sufficient to destroy a limited number of bacteria, and when we keep the number entering a wound within that limit we have achieved practical asepsis.

The essential difference between antiseptic and aseptic surgery is that in the former the germs have supposedly gained entrance to the wound and an effort is made to destroy them there by chemical antiseptics, whereas in the latter we

endeavor to prevent their entrance into the wound. Most surgeons practise both aseptic and antiseptic surgery, but the nearer we get to asepsis the nearer we are to perfection. It is certainly better to prevent the entrance of germs into a wound than to destroy them there, leaving their dead or possibly only inhibited bodies. The chemical solutions used as antiseptics interfere with union and may cause poisoning. When the wound is an accidental one and not of the surgeon's making, it must be treated antiseptically, and in preparing for an aseptic operation more or less antiseptics are used. The chief danger in practising antiseptic surgery is that the surgeon may place too much dependence upon his chemicals and be careless about the entrance of bacteria into the wound. Present-day operative surgery demands that whatever of antisepsis is practised should be before the operation and that asepsis be maintained during and after the operation. It has been clearly demonstrated that chemicals introduced into a clean wound do no good, but do some harm. There is no longer room for controversy as to the comparative merits of antiseptic and aseptic surgery, because at the present time the term used is not applied so much to methods as to results. A sterile wound is an aseptic wound, and a surgeon who secures such wounds is practising aseptic surgery, no matter how he brings it about. Some English surgeons are quite pessimistic about the future of wound healing, because we are drifting farther from the original antiseptic methods, but the fact is that the percentage of aseptic wounds is greater now than at any period in the history of surgery. No matter what method we employ, the principles are those taught us by Lister.

INFECTION.

When bacteria sufficient in number or virulence to overcome the natural resisting powers of the tissues have gained entrance to a wound, it is infected. The evidences of infection are inflammation, suppuration, and a whole train of accompanying symptoms only too familiar to every surgeon.

It is no longer necessary to state, when reporting a surgical operation, that "every antiseptic precaution was observed," because the necessity for these precautions is so well understood by the laity as well as the profession that any one undertaking an operation without them might be held guilty of malpractice. It is just as important to observe every precaution during the operation as before it, for then nature's protection, the skin or mucous membrane, is broken, making an entrance way for the ubiquitous germ. It is in these preparations and precautions that the operator shows his surgical training. One without proper training is prone to feel that, having thoroughly prepared himself and his patient, he has performed his whole duty, and is liable to be careless during the operation. Proper preparation of surgeon and patient means sterilization, and after sterilization every source of contamination must be avoided. No matter how perfect an operating-room, it contains some things that are not sterile, and

contact of the surgeon's hands, instruments, ligatures, and dressings with these unsterilized things means contamination. In order thoroughly to appreciate the dangers of contamination from these seemingly harmless sources, a knowledge of bacteriology is absolutely necessary. The surgeon should be familiar with the different varieties of bacteria and their distribution in the outer world. He should know their ways of entrance into and exit from the body and the conditions which favor their development in a wound. It is only with this knowledge that he can rationally guard against their entrance and development. Every surgeon cannot be a bacteriologist any more than every bacteriologist can be a surgeon, but no man can be a thorough surgeon without a practical knowledge of bacteriology. To such a surgeon the contact of a sterilized hand with an unsterilized object is apt to mean the transfer of pathogenic bacteria from this object to the hand. The prepared surgeon should never touch any part of the patient which is not sterile. He must not put his hands into his pockets, scratch his head, stroke his beard, or blow his nose; he must not handle his eyeglasses nor pick things from the floor. One who habitually makes these mistakes shows lack of either surgical training or surgical sense. It is men of this type who are constantly having suppurating wounds and stitch abscesses, for which they usually hold the nurses, the assistants, or the catgut responsible. These tangible surroundings are the fruitful sources of infection, against which the surgeon must be constantly on guard. In every hospital, in spite of every known precaution, cycles of infection occur, in which a number of cases are involved in close succession. This can sometimes be traced to one septic case which has acted as a focus, but many times the source is never discovered. When these infections occur in the work of one individual, he is septic, and if he cannot become sterile he should quit work for a time. When the trouble is general there is a weak spot in this respect somewhere in the institution, and the only way to overcome it is for every one, from the surgeon down to the humblest nurse who has anything to do with preparation or sterilization, to assume that he or she is individually responsible for the trouble and to act accordingly. It is possible that these infections are from intangible sources, but our present knowledge of infections demands that we act as if they were due to overlooked tangible sources. These cycles will often be traced to the misdeeds of some one who does not understand the principles involved.

Portals of Entrance and Sources of Bacteria in Surgical Infections.

Certain bacteria have their habitat in the human body—for example, the colon bacillus in the intestines and the white staphylococcus of Welch in the deeper layers of the epidermis—and it is these bacteria which make auto-infection possible. It is not definitely known how all bacteria are eliminated from the body, but a natural inference is that the excretory organs have this as one

of their many functions; hence the importance, for an operation, of a preparatory course of treatment which shall carefully regulate these organs. Bacteria cannot gain entrance into the body directly through the skin, but may enter through the numerous natural openings, such as hair follicles and sebaceous glands. Recent observations teach that the sweat glands rarely afford an entrance way for bacteria, and that the danger of infection from sweating hands has been greatly overestimated. Harrington made repeated examinations of sweat obtained from sterilized hands and arms and placed in heated sterile glass cylinders, and in every instance found it sterile. The sweat dropping from an unsterilized face or beard is, of course, an entirely different matter.

The most common entrance way of bacteria is through a wound in the skin. The danger of infection through a wound is not in proportion to its size, the smaller wound often being the more dangerous. In operative surgery the great danger is that bacteria may be introduced into the wound by dirty hands, instruments, ligatures, or dressings. The surgeon and his co-workers are the guards whose duty it is to prevent infection, and the various antiseptics and methods of sterilization are their weapons. Before the days of Lister the surgeon was not responsible for wound healing, but now "the fate of the wound rests in the hands of the one who applies the first dressing." Flies and other insects may infect a wound by carrying bacteria or through their excreta. Pyogenic staphylococci have been found in the excrements of flies. There are, therefore, more potent reasons for excluding flies from hospitals than the comfort of the patients.

Since it has been demonstrated that Lister laid undue stress upon infection through the air, it is quite possible we have gone to the other extreme and do not pay enough attention to this possible source of infection. Just how great the dangers of infection through the air are is still a matter of controversy. Bacteriologists and surgeons are somewhat at variance in this matter, for while the former can demonstrate the presence of bacteria in the atmosphere of every operating-room, the latter can show a long series of aseptic wounds without any special precautions having been taken against air infection. Sea air, where there is no dust, and sometimes high mountain air are free from bacteria, but they are always present in air near the ground where there is dust. They are more numerous in the city than in the country, and are often more numerous in hospital wards than elsewhere. The fact that aseptic work can be done in a germ-laden atmosphere proves that the number of pathogenic bacteria carried in that way is not so great as to overcome the natural resisting powers of the tissues. Indirectly, however, the air is a dangerous source of infection. Bacteria are carried on particles of dust, which have a natural tendency to settle and accumulate on tangible objects, which may cause contact infection. Wounds should be protected from a dust-laden atmosphere. This can be accomplished by moisture, which encourages the natural tendency of the dust to settle.

All manner of bacteria are constantly found in the mouth, nose, and throat; so it is only rational to conclude that they may be carried into a wound by the breath of the surgeon or his assistants. It has been maintained that air is sterilized as it passes through the lungs, and that the lungs are free from bacteria. Even if this be true, air becomes contaminated on passing through the mouth, for breathing into culture media will almost invariably yield cultures. One hospital bacteriologist, after having the internes in his hospital breathe into media several times, was able to tell, from the character of the cultures, which interne had breathed into a certain medium. No one with a sore throat should be allowed to come near a wound. It has been demonstrated by careful observers that in ordinary conversation minute droplets of saliva are being constantly thrown out in all directions, and these same observers have proven that saliva may be richer in bacteria than the foulest sewage. It would seem, in face of these facts, that a surgeon who would, as nearly as possible, remove every source of infection, must either refrain from talking into the wound or wear a mask. The chances of infection through the breath must be very remote, because the moist surface of the nose and mouth will surely capture most of the bacteria, but the dangers from the saliva are well worth considering.

Conditions Favorable to the Development of Infection in Wounds.

Bacteria are plants, and like all other plants require certain conditions for their growth and development. Unfortunately, a wound affords many of these conditions. There are, in a wound favorable to the development of bacteria, many general underlying conditions for which the surgeon is in no way responsible; and, since it is impossible for him to prevent the entrance of some germs into the wound, it is his duty to remove or prevent every condition favorable to their development that he possibly can. Every operation is said to be an experiment in bacteriology and pathology, but we have learned that a wound infection is a vastly different affair from the artificial infection of a culture medium in a laboratory; because the living tissues furnish so many complex conditions, both against and in favor of the development of bacteria, that the results following their entrance into a wound cannot be definitely foretold. The surgeon cannot solve problems in bacteriology without a bacteriologist, but the fact that his culture field is a wound in living tissues enables him to discover some clinical bacteriologic facts that cannot be discovered through a dead medium. This accounts for the occasional difference between the bacteriologist and the surgeon. It must be admitted, however, that most of these differences are explained by the fact that the surgeon too often indulges in *post-hoc* reasoning and attributes certain clinical results to something he has done or used which really had no bearing in the matter.

There are inherent in individuals many conditions that favor the development of infections—conditions that are spoken of as predispositions. These conditions are not well enough understood to enable us to draw many practical conclusions concerning them, and the more we learn of the habits of bacteria the less stress we lay upon predispositions, because, the local conditions being favorable, bacteria will develop in all ages and conditions of human beings. It is well understood that the extremes of age, anæmia, diabetes, and Bright's disease are favorable to the development of infections, presumably from a diminution of the natural resisting powers of the tissues under these conditions.

There is a great difference in the natural virulence of pathogenic bacteria, the colon bacillus and the streptococcus being respectively fair representatives of the mildest and most virulent of the pyogenic micro-organisms. The number of bacteria entering a wound doubtless has a decided bearing upon the early history of an infection, but the natural tendency is for them to multiply so rapidly that this is probably not a lasting factor. The source of an infection has a very decided bearing upon its virulence. Pathologists teach that the virulence of bacteria depends largely upon their ability to form toxic products through which they produce their pathogenic effects. The extreme virulence of infections coming directly from the human body is doubtless due to the recently acquired habits of the bacteria, and to the fact that toxic products are introduced with them.

Tissues vary somewhat in their susceptibilities to infection and in the pathological changes that result from infection. The dense, highly vascular tissues, such as the lips, cheeks, and tongue, have so great a resisting power as to have often brought aseptic technique into considerable disrepute in the minds of those already disposed to question its value, because primary union will usually take place in these tissues even when aseptic precautions have been very scant or inefficient. On the other hand, synovial membranes, the medullary tissue of bone, and adipose and loose connective tissue are exceptionally susceptible to pyogenic infection. Normal mucous membranes are very resistant to microbic invasion, but a paralyzed bladder is very easily infected. Their exceptional resisting power is very helpful to the surgeon, because it is almost impossible to sterilize a mucous membrane when preparing for an operation. There is probably no greater contrast in this respect than that between the peritoneum and joint surfaces. The peritoneum was considered by old-time surgeons their greatest enemy, because their wounds were all septic, and a traumatic septic peritonitis is practically a hopeless condition. With our present knowledge of infection, however, we know that the peritoneum is the surgeon's greatest friend, because of its wonderful ability to care for itself. Joint surfaces have a very low resisting power and are very liable to infection. No one who is uncertain of his asepsis should open a joint, because he is almost certain to have an infection. The older surgeons recognized the dangers of an infection of a joint,

but they did not fear it as they did peritoneal infection, because it is possible to drain an infected joint and restore the patient to health, and sometimes to preserve the function of the joint.

A ragged wound caused by injury or rough handling and dissecting is better soil for the growth of bacteria than a clean cut. Blunt dissections and too heavy or prolonged pressure by retractors are to be avoided. Necrotic tissue from any source offers food for germs. Masses of tissue should not be strangulated by ligatures or forceps. Ligatures and sutures must never be tied tighter than is absolutely necessary. One serious objection to chemicals in a wound is that they are liable to cause necrosis of tissues, thus inviting infection. Foreign bodies, such as drainage tubes and unabsorbable ligatures, interfere mechanically with wound healing and encourage bacterial growth. Drainage tubes cannot be dispensed with altogether, but should always be considered a necessary evil. Many surgeons still use silk for buried sutures and get good results, except in a certain percentage of cases in which they cause abscesses and fistulæ; but one who has become familiar with the use of catgut is never content with silk. Tension in a wound from any cause encourages bacterial development by interfering with the circulation and thus reducing the resisting power of the tissues. Careful hæmostasis is a very essential part of aseptic surgery. Hemorrhage in a wound causes tension and interferes mechanically with healing by keeping the tissues apart. Bactericidal properties have been attributed to living blood and blood serum, but dead blood affords nourishment for bacteria. Blood serum is being used constantly as a culture medium in laboratories. It is an open question whether the bactericidal properties attributed to blood serum are not due to living cells known to be the enemies of bacteria. Be this as it may, all practical surgeons agree that a blood clot in a wound is an element of danger to be avoided whenever possible. There are times, however, when dead space is unavoidable, and then a blood clot is our best resource. In the soft tissues no artificial substance has been found so satisfactory for filling as blood clot, and until a very recent date Schede's method of filling bone cavities with aseptic blood clot was the best method known. Of late, however, Mosetig has given us an artificial filling which is better. Every effort should be made by the surgeon closely to approximate the tissues so as to leave no dead space to be filled with the body fluids. When dead space cannot be overcome without undue pressure from bandages or sutures, it is safer to depend upon nature to fill the cavities.

DISINFECTION AND STERILIZATION.

Disinfection and sterilization are the terms applied to the various methods of destroying micro-organisms and rendering sterile everything pertaining to a wound. The means employed are mechanical, chemical, and dry or moist heat.

The most reliable agent is moist heat through steam. Absolute sterilization of instruments, dressings, and ligatures is possible by heat, but absolute sterilization of hands and skin is impossible, because heat of sufficient intensity cannot be employed, and we are obliged to depend upon mechanical and chemical agents.

Skin Sterilization.

Volumes have been written on the subject of skin disinfection during the evolution of aseptic surgery, and the end is not yet and possibly never will be, because perfection would seem to be unattainable. Practical sterilization, however, is attainable, and the more exacting our methods to achieve it the nearer we approach perfection. There is no one accepted method, but there are many good ones. It is not so much the method chosen as the conscientiousness with which the chosen method is carried out, which leads to results.

Mechanical Purification.

American surgeons are practically unanimous in their opinion that mechanical cleansing by means of soap and brush is the most essential and reliable means of skin disinfection. This is usually supplemented by chemical aids, but the amount of chemicals used has gradually grown less and less, so that at the present time many surgeons place very little dependence upon them. The skin cannot be properly disinfected by chemicals alone. Thorough scrubbing will under ordinary circumstances render them superfluous, but they are still considered a valuable accessory by most surgeons. Scrubbing is so often inefficiently performed that it is wiser to employ some chemical accessory as a routine. A surgeon should take good care of his hands at all times. He should not do anything that will make them hard and rough, because this diminishes their tactile sense and makes them more difficult to disinfect. Kocher said that the time when a surgeon should wear gloves is when he is not operating. He should not put his hands into pus or dress septic wounds without gloves, for, in spite of the most careful efforts at sterilization, the Staphylococcus aureus has been found on a surgeon's hands for several days after handling septic cases. For mechanical purification, soap, hot water, brushes, and nail cleaners are necessary. Running hot water is a great advantage and should be provided in every operating-room. There should be foot attachments, so that the surgeon need never touch the faucet with his hands. When basins are used, the water in them should be frequently changed by an assistant. The running water, besides being clean, aids mechanically by carrying off the loosened and dissolved dirt.

Soap made by heat is usually free from bacteria, but that used in operating-rooms should be resterilized. Soap dissolves fat and particles of dirt so that they can be more readily removed by friction with gauze and brushes. In an emergency any good soap may be used, but soaps impregnated with chemicals have

proven unsatisfactory. The green soap of the pharmacopœia is the best and is that in most common use. It may be used as it is found in drug stores, dissolved in water, or in the form of tincture. The supply furnished for the preparation of hands and skin should be protected against infection. This matter is too often neglected. An open soap-dish so located that dirty hands and brushes can be dipped into it and dirty water splashed into it is a source of danger. Hands at the beginning of preparation or in the midst of an operation on a pus case are septic, and should not be dipped into the soap any more than they should be allowed to touch the sterile dressings. No matter how clean a brush may be, it cannot remain so after being rubbed over a dirty, possibly pus-covered hand. Soap containers can now be had with a valve in the bottom, which when pushed up will allow the liquid soap to flow into the hand. These can be attached to the wall at a convenient spot over the wash sink. Liquid soap can be put into a wide-mouthed sterile bottle, and the mouth then covered with sterile gauze, through which the soap can be poured.

Brushes cannot be sterilized by chemicals, but can be made sterile by boiling them for five minutes. A longer boiling is unnecessary and soon destroys the brush. After every operation the brushes should be thoroughly washed so as to remove soap and gross dirt, and then boiled. They should then be kept in a 1 to 1,000 bichloride solution ready for use. A hog bristle brush of medium stiffness is the best. Too stiff a brush injures the skin and is uncomfortable, and too soft a brush will not remove the dirt. The wood fibre brush is cheap and bears boiling better than the bristle brush, but it is too stiff for comfort. It is folly to expect one brush to clean several pairs of hands without reboiling. Each surgeon and assistant should have at least two brushes. With the first he should loosen and remove all gross dirt, after which he can finish the disinfection with the second. The brushes should be used freely on the hands, especially on the palms and about the nails, for five or ten minutes. For the arms and other parts of the body where the skin is soft, a vigorous scrubbing with soap and a piece of gauze is better, as the brush is liable to cause abrasions or erythema.

Hand scrubbing, in order to be thorough, must be systematic, for otherwise some parts may be untouched. First, the palms of the hands and fingers should be gone over thoroughly, then the skin between the fingers, next the nails and dorsum, each finger being scrubbed separately, and so on until every nook and corner is thoroughly cleansed. This process should be repeated continuously for ten minutes. A right-handed person is prone to devote too large a portion of the allotted time to his left hand, because it is easier for him. A nail cleaner should be used before, and another sterile one several times during, the scrubbing. All hangnails should be removed and abrasions touched with ninety-five-per-cent carbolic acid.

The skin at the seat of operation is much less likely to be a source of deep wound infection than the surgeon's hands, but should be just as carefully pre-

pared. The habitat of the Staphylococcus albus of Welch is in the deeper layers of the epidermis, and, because of its inaccessibility, is a frequent source of stitch abscesses and mild superficial infection in spite of most careful preparation. The seat of operation should be carefully shaved the day before the operation and thoroughly scrubbed. It is customary in most hospitals to apply an antiseptic dressing at this time, to be left over night, but this is not absolutely necessary. Any wet or irritating application is objectionable because it interferes with the patient's rest. The only advantage of this procedure is that less time will be required to prepare the patient on the operating-table. No matter how well the parts have been prepared beforehand, they should be cleansed again just before the operation. It should be remembered that it is quite possible to overdo the scrubbing. It is a mistake to set up an erythema by overvigorous scrubbing, by the use of too rough a brush or towel, or by irritating applications. The skin should not look like a boiled lobster, but should have a healthy pink glow. Mustard, corn meal, and other substances having no bactericidal properties are used by some surgeons for hand cleaning, but they act only mechanically and are inferior to soap and brush.

Irrigation at one time occupied a very important place in surgical technique, solutions of various kinds being run into wounds and cavities of all kinds, and at that time it was doubtless necessary; but now irrigation is employed only occasionally when specially indicated, and is a mechanical rather than a chemical process. A fresh sterile wound should never be irrigated, because it does no good and may do harm. The dry technique yields better results and is much more comfortable for surgeons and assistants, who at one time were obliged to wear rubber boots or have wet feet at every operation. Even septic wounds should not be irrigated with strong solutions, because they are unnecessary, harmful, and at times dangerous. It may be necessary occasionally to wash out pus cavities when they are too deep to be wiped out with gauze, and in that event sterile water or normal salt solution should be used. Some surgeons irrigate the peritoneal cavity when it contains pus or bowel contents, but the majority feel that it is better to wipe out with gauze than to take the risk of washing the septic material into new territory. When irrigation is used here it should be with sterile normal salt solution at from 110° to 118° F. In operations for ectopic pregnancy after rupture, irrigation with salt solution should be employed after the bulk of the blood has been removed with hands and gauze, after which the abdomen should be left full of the solution.

The normal or physiologic salt solution is so called because it corresponds very closely in specific gravity to the blood. It is prepared by dissolving 6 gm. (ʒiss.) of sodium chloride in each litre (℥xxxiiss.) of water. This is filtered and sterilized by boiling or by placing in flasks in a steam sterilizer, and kept in sterile bottles plugged with cotton. The irrigator should be made of glass or porcelain ware, and attached to a stand which permits of elevating or lowering, so

as to get the required pressure. To the irrigator is attached a rubber tube long enough to carry the water the desired distance. At the end of the rubber tubing should be a glass nozzle, which can be changed and boiled. A rubber fountain syringe makes a good irrigator on a small scale, because the whole apparatus can be boiled. For the abdominal cavity a pitcher is often better than an irrigator, and especially is this true when time is precious. (See Fig. 273.)

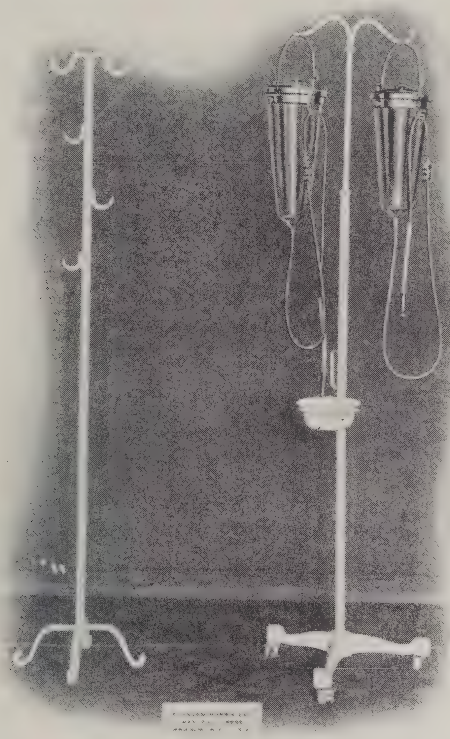


FIG. A.—Irrigator-Stands. The one to the right is adjustable.

In performing plastic operations irrigation can be employed to great advantage, a warm normal salt solution being used in lieu of sponges to keep the parts free from blood, so that the operator can see what he is doing.

Chemical disinfectants still play a part, although a minor one, in skin disinfection. An ideal chemical disinfectant for the skin is yet to be found. Those of sufficient strength to destroy bacteria quickly are too irritating or too dangerous to use in strong solution, and dilute solutions act too slowly to be of great practical value. It is the custom of most surgeons to use a chemical solution after the mechanical cleansing is complete, although there are surgeons whose sensitive hands will not permit the use of chemicals, but who, nevertheless, are able to secure aseptic wounds after mechanical disinfection alone. Elaborate

irrigating apparatus containing a variety of chemical solutions is not much in evidence now. A chemical solution should never be put into a presumably sterile wound.

Bichloride of mercury holds first place in America as a chemical disinfectant, and is commonly used, in aqueous solution of an average strength of 1 to 2,000, in preparing hands and skin. This drug has no penetrating power; hence its action is limited to the surface, where it will kill pyogenic bacteria if brought in contact with them long enough. Harrington has shown that it requires a ten-minutes' exposure of a 1-to-1,000 bichloride solution to kill the Staphylococcus albus, and that weaker solutions require a proportionately longer time. The natural inference is that, from the bacteriologist's standpoint, our bichloride solution as ordinarily used is of little or no value. From the surgeon's standpoint, however, the fact remains that better results have been secured when the solution has been used than by scrubbing alone. The advantages of this chemical are: that it is the most powerful bactericide at our command and that it is freely soluble in water and alcohol. The disadvantages are: that it is so highly poisonous and irritating that strong enough solutions cannot be used to destroy bacteria within a reasonable period of time; that it forms an insoluble, inert albuminate; and that it corrodes instruments very quickly. Notwithstanding these serious objections, its popularity is well deserved, because the skin is more frequently sterile when scrubbing with soap and water is followed by scrubbing in a bichloride solution than when the latter is not used. It kills many germs, inhibits others, and buries many under an impenetrable layer of albuminates, thus rendering them harmless. An alcoholic solution acts more powerfully than an aqueous one. Fuerbringer's method of skin sterilization is one of the best and most popular. After a thorough scrubbing with soap, brush, and hot water, he next scrubs with eighty-per-cent alcohol for one minute, and finally with a bichloride solution. After a long series of experiments Harrington found that a solution composed of commercial alcohol 640 c.c., hydrochloric acid 60 c.c., water 300 c.c., corrosive sublimate 0.8 gm., killed the bacteria in pus from a carbuncle in less than thirty seconds. Hands kept in this solution for two minutes were usually sterile. This solution has been found very valuable in septic cases and for sterilization of hands that have recently been exposed to pus, but, unfortunately, it is too irritating to be employed as a routine procedure.

Carbolic acid was the agent upon which Lister placed the greatest dependence in his early work, but it is now seldom used in the preparation of hands or skin, because of its inferior germicidal powers, its poisonous properties, and its bad odor. Of late years, however, it has come into very general use in sterilizing septic wounds and cavities. For this purpose the commercial ninety-five-per-cent solution is used in its full strength. Powell taught us that it can be applied freely in this strength to the skin or a wound if followed within two minutes

with alcohol, which acts as an antidote. This strong acid immediately hardens the tissues so that they cannot absorb it quickly. A much weaker solution would be dangerous, because it would not harden the tissues. A septic suppurating surface or sinus can be so thoroughly prepared by using the pure carbolic acid in this way that an aseptic operation may be performed upon the part. In the case of old bone cavities successful results can be obtained by clearing out the sequestra, pus, and detritus, and then, after a thorough sterilization with the pure carbolic acid and alcohol, filling the cavity with the Mosetig-Moorhoff bone paste and closing the wound tight.

Lysol is a saponified phenol and a very good disinfectant in an aqueous solution of a strength of from one to three per cent, and is especially well adapted for use in sterilizing mucous membranes. It is smooth and pleasant to the touch, but has a disagreeable odor, which will remain on the surgeon's hands for a day.

Potassium Permanganate and Oxalic Acid.—The Schatz method has been extensively used in this country, notably at the Johns Hopkins Hospital. The hands and arms are first scrubbed for ten minutes with soap and hot running water or many changes of water. They are next immersed for two minutes in a saturated solution of potassium permanganate, after which they are washed in a warm saturated solution of oxalic acid until the color disappears. They are then washed in a sterilized lime solution to remove the excess of oxalic acid, and, finally, they are washed in sterilized water. Robb recommends that after this the hands be washed in a bichloride solution; but few hands will stand so much and so many chemicals, and, since it has been demonstrated that the scrubbing is the essential feature, the more elaborate method seems useless. Kelly's experiments showed that the oxalic acid is the more active sterilizing agent. Harrington has demonstrated that potassium permanganate is a very feeble germicide; that it stains the skin by precipitating a lower oxide, oxygen having been given up to the epidermic scales and other organic matter; but that as soon as one layer of oxide is removed by dissolving in oxalic acid, other layers, indefinite in number, can be formed by a reapplication of the permanganate, showing that the oxidizing process is a very superficial one. He concludes that "a dirty hand may be stained and decolorized as well as a clean one, but the dirt remains. Permanganate removes no dirt and destroys only weakly resistant bacteria." It would seem, therefore, that the real virtue in the Schatz method is that it involves a great deal of scrubbing, which might as well be done without the chemicals.

Iodine is a prime favorite with some surgeons, and has the great advantage of being comparatively harmless. A solution of a deep sherry color is recommended. The iodine method of sterilizing catgut is the most common and one of the best.

Alcohol is not a very active antiseptic, but is a very valuable accessory in hand and skin disinfection. A seventy-per-cent solution is better than the com-

mercial alcohol. It helps to cleanse the skin and hardens it. Nothing a surgeon does in the way of skin sterilization gives him such a sense of cleanliness as washing in alcohol, and this is doubtless the reason why it is so popular. Many surgeons depend on mechanical cleansing followed by the use of seventy-per-cent alcohol, and they secure as good results as are secured by any other method.

Ether is of no value as an antiseptic, but acts as a very valuable adjuvant in skin sterilization by dissolving fat and dirt.

Peroxide of hydrogen has very little value as an antiseptic, but is a powerful oxidizer and is very helpful in cleansing suppurating wounds and deep sinuses. It is capable of doing great harm, however, when injected into cavities to which there is not a free outlet, for it is very liable to force bacteria and toxic materials deeper into the tissues. For cleansing an infected wound it can be used pure or with the addition of an equal amount of water. When used too frequently it interferes with the healing of the wound. When injected into a wound it effervesces and acts mechanically by dislodging particles of dirt and necrotic tissue. This mechanical action probably constitutes its greatest virtue.

Conclusions Concerning Hand Sterilization.

Systematic scrubbing with green soap, running hot water, and brush for ten minutes, followed by the use of a 1-to-2,000 bichloride solution or seventy-per-cent alcohol, or both, will yield as good or better results than are obtainable by any other method. When hands have recently been in or about a septic wound, Harrington's solution should be used.

Conclusions Concerning Skin Sterilization.

The danger of infection from the patient's skin is much less than that from the surgeon's hands. When practicable, the seat of operation should be shaved and scrubbed the day before the operation, although this is not imperative. The scrubbing should be followed by the application of ether and seventy-per-cent alcohol. A sterile dressing should then be applied. Just before the operation the skin should be rescrubbed with soap and gauze, washed with ether, and finally with seventy-per-cent alcohol or a bichloride solution, or both. These methods are quite universally adopted and will yield as good results as any.

Disinfection of mucous membranes is somewhat more difficult than skin disinfection, and, on account of their great liability to be absorbed, chemicals cannot be very freely used. The vagina should be scrubbed and irrigated with a three-per-cent lysol solution some time before the operation, and just before the operation it should be scrubbed for five minutes with green soap and gauze, followed by an application of lysol. Bichloride should not be used. The rectum should be prepared in the same manner, the bowels having been thoroughly emptied the day before.

Rubber gloves are now in quite general use in America, where they were first introduced by Halsted. The objections to them are, that they are somewhat expensive and that they interfere with the surgeon's tactile sense and dexterity. The first objection should be given no consideration, since rubber gloves have undoubtedly saved many lives. The surgeon soon becomes accustomed to their use, so that he can work almost as speedily with as without them. They do delay the operation somewhat by slipping when the tissues are handled and in tying knots, but this loss is compensated for many times over by the additional safety they afford. Mortality rates have been reduced since the introduction of gloves far more than can be accounted for without taking gloves into consideration. This is particularly true among beginners and occasional operators. Every surgeon must be at one time a beginner, and occasional operators are a necessary evil. A thorough-going surgeon can get along without gloves, and many of them do, because they know how to prepare and protect their hands; but every assistant and nurse who has aught to do with the wound, instruments, ligatures, or dressings, should be compelled to wear them. The one argument in their favor which overcomes all possible objections, is that they can be made absolutely sterile and are therefore unquestionably helpful in maintaining asepsis. It is an economy to use only the best quality of seamless gloves made, because they are more pliable, more secure, and last longer. The heavier weight is preferable for much the same reasons, and the surgeon soon becomes so accustomed to them that he does not notice the difference. The wearing of great, clumsy rubber gloves full of holes, and of cotton gloves, is objectionable because they do not fully protect the wound. Gloves should fit so closely so that they will not wrinkle, but not so closely as to be difficult to put on or to constrict the hand when on. After an operation the surgeon should wash all the blood from his gloves before removing them, because when it once dries on them it is very difficult to remove, and, when not removed, may become a source of infection. After this they should be boiled for five minutes, and if not required for immediate use they should be wrapped in a sterile towel and laid away. When cared for in this manner it will be necessary to boil them for only five minutes when they are next wanted. When not cared for properly they will require a much longer boiling, which may do them harm. When gloves are not to be used for some time they should be dusted inside with sterile talc powder to prevent them from sticking together. In hospital work this is unnecessary, provided they are thoroughly dry when put away. A beginner is very liable to tear his gloves when putting them on and off, but one soon becomes so expert that they can be handled quickly and safely. After they have been boiled preparatory to an operation, they should be placed in a basin containing an abundance of sterile water. The surgeon then fills the glove with water, and takes firm hold of the upper end on the palmar side with his thumb inside. He then gently introduces the other hand, alternately extending and flexing

the thumb and little finger until the fingers get well down into the glove, when the latter should be held open and the hand gradually closed, thus pressing out all the surplus water. A glove should not be adjusted with the bare hand, but after both gloves are on they may be adjusted at will without fear of contamination. If the gloved hands are then dipped in a bichloride solution the gloves will not be slippery. It is advised by some that a bichloride solution be used instead of sterile water for putting the gloves on, as an additional antiseptic precaution; but since, as Harrington has demonstrated, sweat from a sterilized hand is aseptic, this is unnecessary and the solution is objectionable because it makes it more difficult to put the gloves on, and the bichloride blackens the finger nails. An elegant way to put on gloves is with a lysol solution, because it makes them smooth, but it is objectionable on account of its disagreeable odor, which remains with the surgeon all day. The hands must be as carefully prepared when gloves are to be used as when they are not, for the gloves are always liable to be torn, and then, if this accident should happen at a time when the enclosed hand had not been adequately sterilized, the patient would be exposed to a corresponding risk of infection. Gloves are in no way a substitute for hand disinfection, but are a very valuable additional protection to both patient and surgeon. Their most valuable use, however, is in septic cases, as they are an invaluable protection to the surgeon at the time and of value to his next patient. An ungloved hand that has been in a septic wound cannot be made safely sterile for many days, but a glove so used can be made absolutely sterile by a few minutes' boiling. The surgeon and assistants should always wear gloves when dressing septic wounds or when examining septic cases. They are of incalculable value in handling specific cases with open sores. If the full list of physicians and surgeons who are annually infected with syphilis for need of this simple precaution were known, it would be startling. Small punctures in gloves may be closed by a rubber cement made for the purpose. Larger tears may be patched with pieces of discarded gloves by the aid of heat. A torn glove finger can be stretched over a test tube, a piece of rubber from an old glove laid over the tear, and then, by running a hot Paquelin cautery around the edge of the patch, the rubber will be made to adhere perfectly.

Sterilization by Heat.

Heat is our most reliable sterilizing agent, and is applied by means of the actual cautery, hot air, hot water, and steam.

The actual cautery has a very limited application as a sterilizing agent, but is occasionally resorted to when a very virulent local infection exists or is suspected. It is very efficient, but the fact that it destroys the tissues limits its usefulness. The same objection unfortunately applies to all forms of heat when applied to tissues.

Hot air or dry heat is very little used as a sterilizing agent because hot water or steam is used instead for almost every purpose for which heat is applicable, and its penetrating power is much greater. Dry heat is used in the Boeckmann process of catgut sterilization with great satisfaction. Aside from this, the chief use made of hot air is in drying dressings and clothing after they have been sterilized by steam.

Hot water is a very efficient sterilizing agent and is almost universally used in the sterilization of instruments, unabsorbable sutures, and gloves. When facilities for steam sterilization are not at hand, utensils and dressings may be sterilized by boiling in water.

Sterilization of Instruments.

Boiling in water is the best agent available for the sterilization of instruments. Cutting instruments, knives, scissors, and needles, however, are dulled by boiling. When they have been washed they should be placed in ninety-five-per-cent carbolic acid for ten minutes, after which they should be kept in sterile water or alcohol during an operation. A square glass or porcelain dish

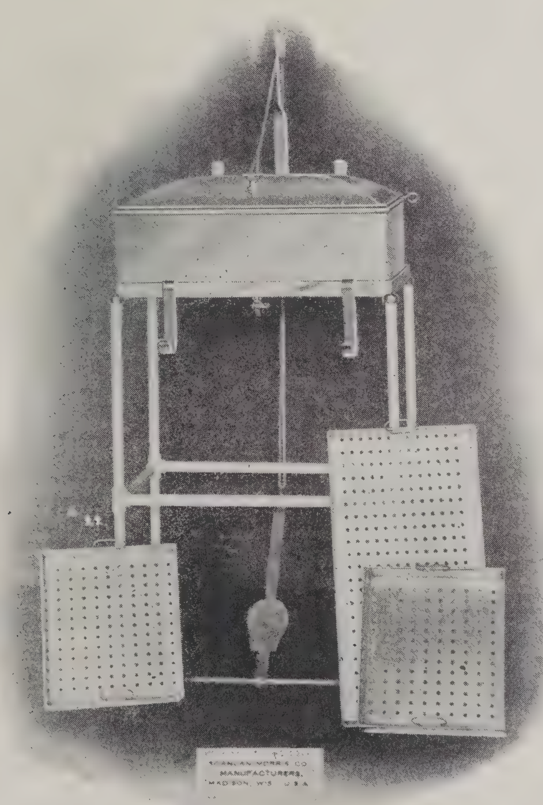


Fig. 274.—Instrument Sterilizer; equipped with one large and two small trays.

should be kept for this purpose; it should be long enough to receive cutting instruments, of such a depth that the instruments are entirely covered by the acid, and provided at one corner with a spout through which the acid can be returned to the bottle in which it is kept. When these instruments are cleaned and washed, after an operation, the ten minutes' immersion will render them absolutely sterile and will not dull them. The other instruments should be boiled for ten minutes. Longer boiling is unnecessary, provided the instruments have been properly washed and scrubbed after the last operation; and, besides, prolonged boiling injures them. To prevent corroding, sufficient sodium carbonate—common soda—is employed to make a one-per-cent solution—which may be roughly estimated at one tablespoonful to a quart. No sterilizing plant is complete without a separate instrument-boiler, which may be heated in the same manner as the other sterilizers (Fig. 274). It is made with a wire basket inside, which enables one to lift at once all the instruments out of the water. Silk, silkworm gut, horsehair, nails, pins, etc., are usually sterilized in the instrument-boiler, either with the instruments or separately. Gloves are sterilized in the same apparatus, but should be boiled separately.

Sterilization of Water.

The difference between ordinary and surgical cleanliness is well illustrated by the fact that water, which is the general cleansing agent in personal cleanliness, is utterly unfit for surgical purposes until it has been sterilized. It can be made sterile by adding chemicals, but they are objectionable and even dangerous. Water can be made sterile by boiling, but it should be filtered as well, because ordinary water, even when sterilized by boiling, contains foreign matter which, in a wound, is objectionable. In an emergency, water can be boiled in an open wash-boiler, from which it may be dipped with a sterilized utensil, but this method affords an opportunity for air and contact infections. Some one may put a dirty hand into it to test the temperature, and the dipper or other utensil used is apt to be laid on a chair, or to be dropped into the boiler after it has been handled with unsterilized hands. The boiled water from any steam boiler is sterile and can be used for sterilizing the hands as it comes from the faucet, but it contains foreign matter, and is therefore not suitable for use in or about a wound. Every hospital should have special apparatus in which water can be filtered and sterilized daily for use in the operating-room. Water-sterilizing outfits, consisting of two reservoirs, one for hot and one for cold water, with a filtering attachment, are now manufactured and answer the required purposes admirably. They are arranged for using oil, gas, or steam (Fig. 275). With a high-pressure outfit, boiling for twenty minutes with a pressure of fifteen pounds is sufficient, for with this pressure the temperature is 250° F., which in that time will destroy all manner of germs. With a

low-pressure outfit the boiling should continue for at least twice as long a time because the temperature is only 212° F. The cold-water tank is so arranged that the water can be cooled very quickly. The outlet faucets when not in use should be wrapped in gauze to keep off the dust.

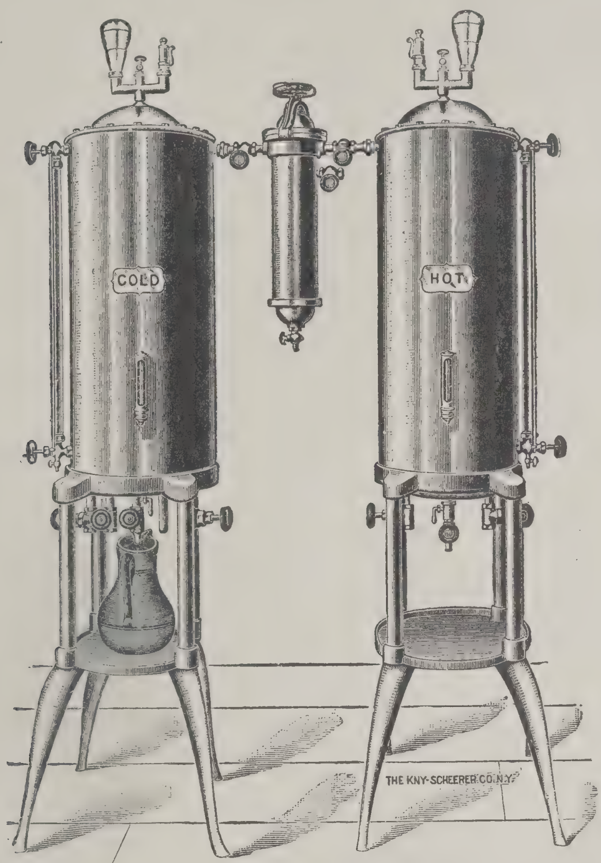


Fig. 275.—Water Sterilizers Arranged for Heating by High-Pressure Steam.

Sterilization of Dressings.

The whole scheme of sterilization is based upon the underlying principles first discovered by Pasteur and first practically applied by Lister. Anything that will destroy bacteria will sterilize dressings. At first, Lister depended upon chemicals almost exclusively, but the objections were found to be so manifold that they have given place to heat in the form of steam almost altogether. For a time manufacturers were kept busy manufacturing the various medicated gauzes, but now, aside from iodoform gauze, which is used in a special class of cases, they are not in the market, because plain sterile gauze has taken their place. Sterile gauze may be bought in sealed packages which are very convenient for those who use it in small quantities. For aseptic

cases the small packages should be used, because when a package is once opened for a dressing it should not be depended upon for later dressings on account of the many possibilities of its becoming infected. It is safer and more economical, however, for every practitioner who does surgical work to have his own sterilizer. Low-pressure sterilizers of small size, arranged for oil or gas, can

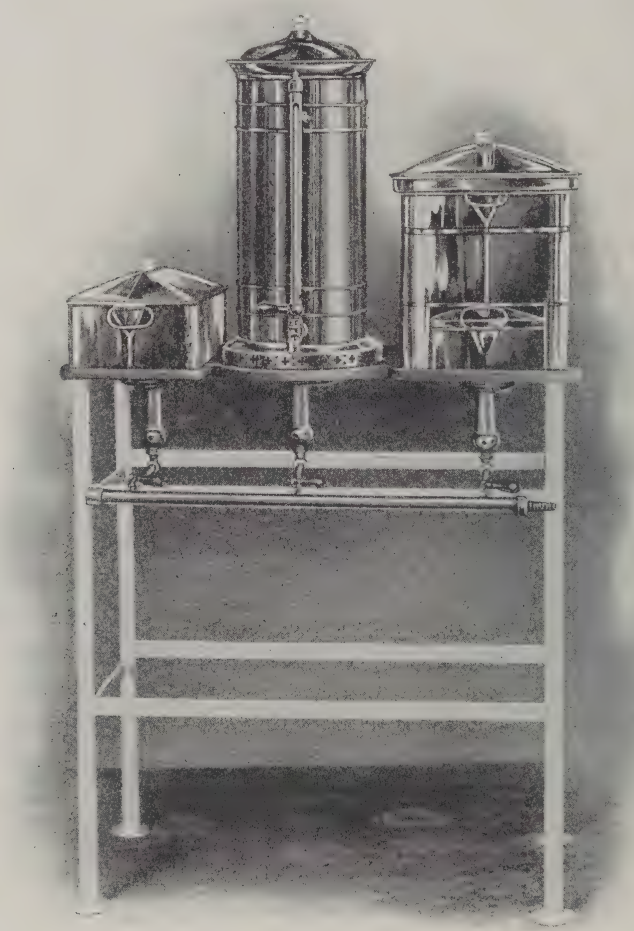
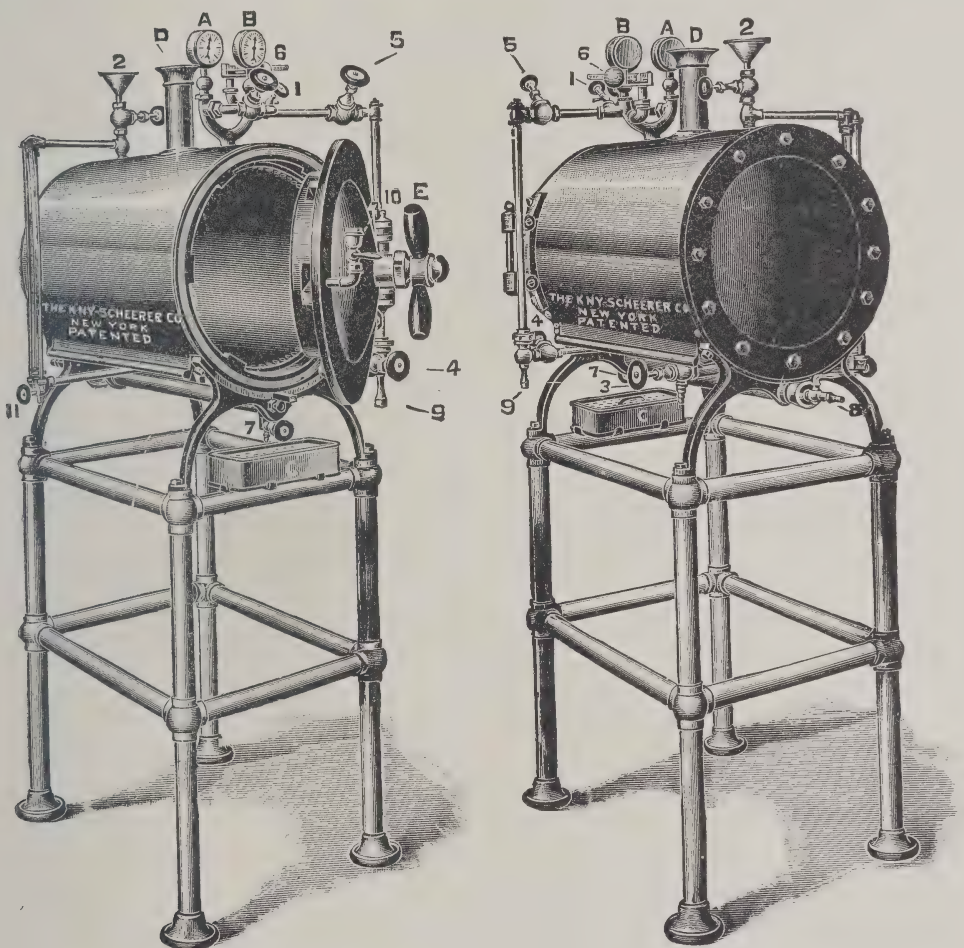


Fig. 276.—Dressing Sterilizer, Instrument Sterilizer, and Water Sterilizer Combined; for office use. (Scanlan-Morris Co., Madison, Wis.)

be bought cheaply and are efficient. The best sterilizing chambers for both water and dressings are made of heavy copper and should be nickel-plated because otherwise the heat discolors the copper (Fig. 276). There are two different varieties of steam sterilizers employed, the high-pressure and the low-pressure. The high-pressure apparatus (Fig. 277) is so arranged that a pressure of from ten to fifteen pounds to the square inch can be gained very quickly

and maintained indefinitely at a temperature of from 240° to 250° F. This high-pressure steam is the most penetrating and has the greatest bactericidal action. This variety of apparatus is arranged for heating by steam from the ordinary boilers of a building, or by means of gas-burners, and is the variety commonly used in large hospitals. When boiler steam is used as a heating medium it should be of not less than thirty-five pounds pressure. In smaller institutions where a low-pressure engine is used or where the house plant is not used at all times, a small boiler especially for the sterilizing outfit can be



Showing Front View. Showing Rear View.

Fig. 277.—The Kny-Scheerer Dressing Sterilizer (1903 Model).

A, Gauge to register pressure within chamber; *B*, gauge to register pressure within jacket; *C*, pan to catch any condensation from chamber; *D*, ventilating pipe for gas combustion; *E*, handle for opening or closing door.

1, Valve for the discharge of air displaced by water; 2, valve and funnel for filling jacket with water; 3, valve for injecting water into jacket; 4, valve connecting air ejector to chamber; 5, valve for controlling steam for vacuum apparatus; 6, valve for admitting steam from jacket to chamber; 7, valve for withdrawing any condensation from chamber; 8, gas attachment; 9, ejector or vacuum apparatus; 10, air filter for destroying vacuum; 11, valve to empty water from jacket.

used conveniently and economically. High-pressure steam is efficient. The objections to it are that it is expensive and requires skill to handle it properly. A very essential part of this apparatus is the vacuum or air-exhaust attachment, by means of which the air is withdrawn from the sterilizing chamber before steam is turned in. Without this feature the temperature will vary in different parts of the chamber and the degree of temperature attained will be uncertain. Unskilled persons are liable to turn the steam into the sterilizing chamber

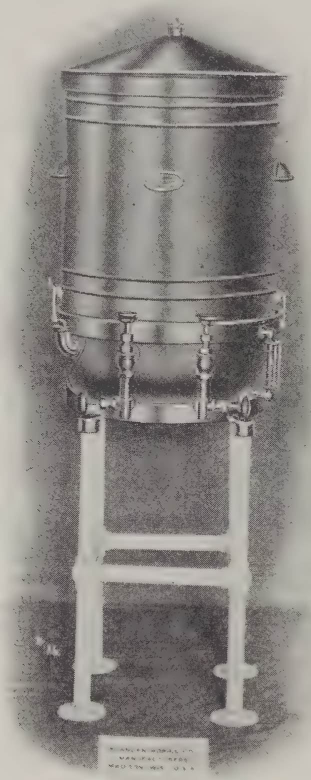


Fig. 278.—Large Hospital Sterilizer with Steam Coils.

without having emptied it of air, and the result is that the dressings are not sterilized.

The low-pressure sterilizers are the ones commonly employed in physicians' offices and in small hospitals, and are popular in large hospitals in the West (Fig. 278). When properly constructed they are efficient and more economical than the high-pressure sterilizers; furthermore, they require no special skill to operate (Fig. 279). The introduction of the steam from above is an essential feature of the low-pressure sterilizer, for in this way the air is thoroughly driven out. The live or moving steam coming up through a jacket outside

of the dressings equalizes the temperature and prevents condensation. Before the steam is turned into the dressings they should be thoroughly heated by running the hot-air current through them. Either oil, gas, or steam can be used as a means of generating the heat for these sterilizers. When the low-pressure system is used the dressings should be steamed for one hour. Fractional sterilization of dressings is unnecessary, but it is customary in most hospitals to sterilize them the evening before and again in the morning of the day on which the operation is to take place. Cotton, gauze, towels, caps, gowns, etc., can be loosely rolled in cotton cloths kept for the purpose before

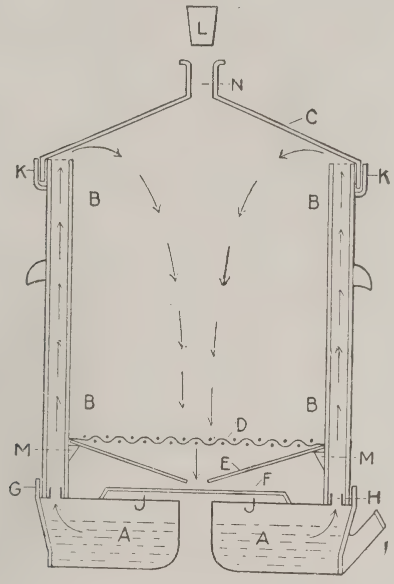


FIG. 279.—Diagram showing Cross-Section of the Large Sterilizer Represented in the Previous Figure. Steam is generated in boiler (*A*) and ascends from the boiler through double walls to the top of the sterilizing chamber, where it is held in suspension by the cover (*C*); then by its own pressure it is forced down through the dressings, excluding all the air from the sterilizing chamber. At the point (*E*) it finds an escape and is dissipated by hot air from the burner, which is placed below. After the process of sterilization is completed, valve (*L*) is thrown open and the steam in the boiler passes up through the double walls and out through this valve. The steam in the sterilizing chamber is driven out by the hot air coming in from the burner below. The hot air circulating up through the dressings thoroughly dries them out so that they may be taken out hot and dry ready for application.

The following is a list of the parts of the Scanlan sterilizer:

(*C*) Lid, (*N*) valve lid, (*KK*) water-tight joint for lid, (*BB*) sterilizing chamber, (*D*) wire tray to support dressings, (*E* and *F*) shields to protect the dressings from being burned, (*AA*) boiler for water, (*I*) point for filling boiler.

being placed in the sterilizer. They must not be rolled or packed too tightly, for this prevents the steam from penetrating them. Tight packing in too small a sterilizer is a common error in sterilization. Utensils for the operating-room—basins, pitchers, etc.—can be sterilized in the same chamber used for the dressings, but it is a great convenience in hospitals to have a separate utensil sterilizer (Fig. 280).

SURGICAL DRESSINGS AND SPONGES.

The most remarkable feature of modern surgical dressings is the simplicity of the dressings and sponges. Before the Listerian era all manner of substances such as jute, bran, and sawdust were employed with what would now be considered unsatisfactory results. Lint made from linen or flax was the most popular. The War of the Rebellion is sometimes spoken of as the era of "wet-rag surgery," because the dressing commonly used then was a cloth wet in cold water. Not long after the close of the war many surgeons tried the treating of wounds without dressings. A great variety of necessarily infected ointments held high place in surgery, and were favorites among the laity. They were usually spread on cloth or surgeon's lint, made for the purpose. With our present knowledge of infection and its dangers we often wonder how wounds ever healed so soon, subjected as they were to so many sources of infection. In antiseptic technique as taught by Lister the dressings were elaborate and complicated, and were impregnated with chemicals. Their use was based upon the belief that air infection was a great danger during and after the operation, but since it has been demonstrated that this is one of the least of the dangers the dressings have assumed their present simple form. Then the chemicals in the dressings often irritated the patient, but now our experience demonstrates that they are entirely unnecessary. The requisites of a surgical dressing are that they shall be sterile, dry, and comfortable. The materials needed for the dressing of an aseptic wound are the coarse, loosely woven cotton cloth manufactured for the purpose and known as gauze, specially prepared cotton known as absorbent cotton, and a cotton roller bandage.

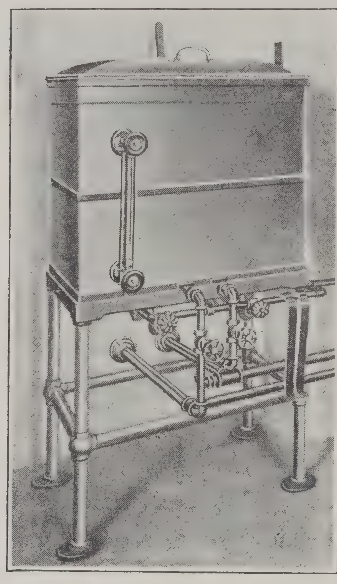


FIG. 280.—Large Hospital Utensil Sterilizer, for use in Sterilizing Wash Basins, Pus Basins, Trays, etc.—(From Scanlan-Morris Co., Madison, Wis.)

Gauze is applied next to the wound and must be sterile so that it will not cause infection. It must be dry and loosely woven so that it will keep the wound dry by quickly absorbing moisture that may come from the wound. Moisture is necessary for the development of bacteria, and a dressing which will keep the wound dry prevents their growth, where a moist one would encourage it. The absorbent cotton should be applied outside the gauze for protection and comfort. It prevents the germs from gaining access to the wound, and by its bulk and softness protects the wound from injury. Absorb-

ent cotton is used in bacteriologic laboratories to protect culture media from air infection. The bandage is applied over dressings to keep them in place. The gauze may be cut into squares of various sizes, according to the dimensions and location of the wound, or it may be cut in long strips of various widths and loosely rolled up. These various-sized pieces of gauze should be made into bundles of moderate dimensions and each bundle wrapped and pinned loosely in a cotton cloth ready for sterilization. They are then piled loosely —not packed—in the sterilizing chambers and sterilized by steam, followed by dry hot air. When removed from the sterilizer the packages should be laid on tables covered with sterile towels. The wrappers should not be removed until the gauze is about to be used. The wrapper is sufficient protection against contamination for a short time, and, when it is removed, should be considered septic and not allowed to touch the gauze again because it has been handled and perhaps otherwise infected. Surgeons' gauze can be purchased by the yard or the bolt from instrument houses or dry-goods houses. In an emergency the common cheese cloth may be used, but it is too closely woven and too hard for general use.

Cotton in its natural state is unfit for surgical dressings because it contains an oily substance which prevents it from absorbing liquids, but, with the development of antiseptic and aseptic technique, manufacturers have prepared it so that it has entirely supplanted linen as a surgical dressing. At the present time cotton fibre is in such great demand for surgical purposes that its preparation has become a great industry. The best quality of surgical cotton, or absorbent cotton, comes from the factory in long strips rolled between layers of tissue paper. When dropped into water it will absorb so rapidly that it sinks almost immediately. A good quality will absorb as high as fifteen times its own weight of water. Only the best grade should be used in surgery; it is manufactured from the fresh cotton fibre, while the cheaper grades are made from cotton rags taken ofttimes from filthy surroundings. Absorbent cotton of good quality may be bought at most drug stores in packages of a size to suit the purchaser. Absorbent cotton must be cut in pieces of various size, wrapped in cotton cloth covers, as gauze is prepared for sterilization, and then sterilized by steam, followed by hot air. The processes of preparation do not sterilize the cotton and it should not be used on an aseptic wound without having been sterilized by steam.

Gauze bandages have almost entirely supplanted bandages made from heavier cotton cloth. They are made from a closer woven gauze than that used for dressings; this gauze, however, is light and elastic. The manufacturers sterilize them and their paper wrapping, thus rendering them fit to be applied outside the gauze and cotton without resterilizing. The use of these elastic bandages over liberal layers of gauze and cotton has practically eliminated the danger of tight bandaging.

Sponges are no longer used in surgery, but they were for a long time considered an essential part of surgical technique. The name sponge is often applied to the gauze pads now employed, and the use of them is termed sponging by some. Gauze pads are not so soft and elegant as marine sponges, but the former can be made sterile by steam, while the latter do not bear heat well in any form, and all methods of sterilizing them have proved disappointing. The pads, or "sponges," for use during the operation are made from the same gauze as that used for dressings and are sterilized in the same manner. The gauze is cut in pieces about six or eight inches square and folded so as to turn all rough edges in and make a pad three or four inches square and four layers thick. All ravellings must be inside so as to prevent them from being left in the wound where they may act as foreign bodies. Each pad is used but once and then thrown away. Pads have two important advantages over sponges: first, they can be made sterile; and second, they are inexpensive and therefore need not be used but once. For abdominal work large gauze pads are made for protecting the bowels and keeping them out of the operator's way. These pads may have tapes attached to them to which forceps can be fastened to prevent the pads from being left in the abdomen. This is unnecessary, however, where the operating-room force is trained to keep careful count of the number used and to see that all are accounted for before closing up the wound. The small pads are better when used dry, but the large ones which are left in contact with the peritoneum for some time should be wrung out of a warm normal salt solution just before using, to prevent them from adhering to and injuring the peritoneum. The pads must be thoroughly sterilized before, and carefully protected from contamination during, the operation, because they come in direct contact with the wound.

Towels, preferably of cotton, are a very necessary and convenient article for use during an operation. They should be of medium size—about eighteen by thirty-six inches,—of medium weight and without fringe. This material may be bought by the bolt and cut into the desired lengths. Since they are to be used more than once, they should be very carefully washed and boiled after each operation, and thoroughly sterilized by steam just before the operation. They are usually sterilized in the same chamber with the gauze. They should be made into bundles, not too large nor too closely packed, and should be covered with a cotton-cloth wrapper just as the gauze is prepared for sterilization, and should be handled and protected in the same manner after removal from the sterilizer. They are used for drying the hands of the surgeon and assistants, and for covering the unsterilized portions of the patient's body to prevent contamination of hands, dressings, instruments, ligatures, and sutures. Sheets of larger size, made of heavy cotton sheeting and prepared in the same manner, may be conveniently used for draping the patient, but towels are indispensable. The towels may be pinned together with sterile safety pins to keep them in

place. They should not be wet in a bichloride solution, because they would then corrode the instruments.

Caps made of cotton or linen should be worn by the surgeon and assistants to prevent infecting agents from falling from the head into the wound. They should be sterilized and cared for in the same manner as the dressings. The surgeon should cover his beard with gauze. He should not wear nose glasses, especially when doing abdominal work, as they may need adjusting during the operation, and they have been known to drop into the abdomen. Spectacles are better and safer.

Fig. 281.—U-shaped Operating Mask, on a Spectacle Frame.

Masks are not worn by all surgeons, but most careful surgeons wear them. They are undoubtedly an additional source of protection, and when the surgeon is obliged to talk during the operation he should wear a mask, for it is impossible to avoid talking into the wound at times; and when he does, droplets of saliva crowded with bacteria are bound to enter the wound unless prevented by a mask. The mask is usually made from a piece of sterile gauze which is tied over the top of the head, coming down over the nose and mouth (Fig. 281). This form of mask is liable to come off or to become displaced so as to interfere with vision. It is better to attach the mask to the cap or to a wire frame worn like a pair of spectacles. The mask should be sterile, and to that end should be changed often. It is better to wear no mask than to wear a soiled one.

Operating gowns are almost universally worn in the operating-room. They may be made of linen or cotton, should fit loosely, and should be of sufficient length to reach the wearer's shoe tops. The sleeves may be long or short according to the fancy of the operator, but the short sleeve which fastens just above the elbow is the one most commonly used. The long sleeve is objectionable because it becomes wet and disagreeable, and when it becomes bloody cannot be changed without changing the whole gown. To continue to wear a gown with bloody sleeves is very like using a sponge many times over and it looks very untidy. The gowns are to be prepared for sterilizing just as the dressings and towels are, each gown being wrapped separately in a cotton cloth

and then sterilized by steam. After being removed from the sterilizer they must be protected as carefully as the dressings, not being touched by unsterile hands. A nurse whose hands are not sterile may unpin the wrapper and open it so that the gown may be unfolded and put on by sterile hands. After the operation, gowns must be carefully washed and boiled. The gown is for the protection of the wound and not of the surgeon or his clothing, and must be as carefully sterilized and handled as the dressings. It is customary for surgeons and assistants to wear rubber-cloth aprons for the protection of their clothing. These aprons should be put on before the hand-scrubbing begins, but the gown should not be worn until just before the operation.

The trained nurse is the one to whom the preparation of all dressings and suture materials is entrusted, and it is therefore very necessary that she be thoroughly trained in the principles underlying their preparation. No matter how honest and faithful she may be, if she is merely following out instructions without understanding the principles involved she is certain to make mistakes. The trained nurse is needed in aseptic surgery. She is often trusted more than the assistant, because a very important part of her work—the preparation work—is done in the absence of the surgeon, and if she were to fail in the performance of her duty the surgeon would surely fail to get aseptic wounds. There is a tendency at the present time to overtrain nurses. An overtrained nurse is more objectionable than an undertrained one because she is disposed to have opinions concerning matters which are none of her concern. It is a mistake to teach nurses pathology, diagnosis, and treatment, because at best they can get only a smattering of knowledge, which is unnecessary, always annoying, and often dangerous. The well-trained nurse is the one upon whom we all depend as an invaluable assistant and adjunct, but we do not need her as a consultant. It is wrong for the surgeon to place too much responsibility upon the nurse during the after-treatment. He himself should carry that to the end. Doctors who have not had proper training and who undertake to do surgery are prone to blame the nurse for their failure in asepsis, but the average trained nurse is a much safer person in the operating-room than a poorly trained doctor.

Dusting powders, at one time considered so essential, are seldom used now save from force of habit. They have little or no value as disinfectants. Their only real value is in keeping the wound dry, and this can be done much better by dry sterile gauze. When a wound is dried by a powder it forms a scab and prevents the escape of discharges instead of draining them away. Bacteria often thrive when surrounded by the powders in use. Aseptic wounds do not need powder, and their value in septic cases is questionable.

Iodoform at one time was so universally employed in wound dressing that a surgeon might be recognized anywhere by the disagreeable odor. The extremes to which its use was carried are being followed by the usual reaction, and this

drug may be altogether neglected after a time, regardless of its merits. Not many would think of dusting a fresh wound with iodoform now, and only few use it in septic cases. Bacteriologists demonstrated that it is not a germicide and that it is necessary to sterilize it when injected into cavities. It is to be noted, however, that many of those who were enthusiastic in its use seldom employ it now. Iodoform is still used quite extensively in the form of an emulsion and of iodoform gauze, but even they do not occupy the high place they once did. They are used most frequently in local tuberculoses because of the supposed inhibitory action of iodoform upon the tubercle bacillus. The Mosetig-Moorhoff bone plug, so lately of prominence, contains a large percentage of iodoform.

The iodoform emulsion commonly used consists of ten per cent of iodoform in olive oil. This has been given a very extensive trial by orthopedists as an injection in tuberculous joint disease, but has been practically abandoned because it is disappointing. It is still employed in injecting cold abscesses. The abscess is first emptied through a small incision and irrigated with a normal salt solution which acts mechanically, forcing out the detritus often found in these abscesses. It is dangerous and unnecessary to irrigate a large abscess of this kind with a bichloride solution. In making the incision in these cases the skin should be drawn to one side so that it and the deeper tissues are opened at different points, making a valve-like opening. The emulsion should be warmed and thoroughly shaken, when it can be injected into the cavity by a sterile glass or hard-rubber syringe. Several ounces may be injected with safety, since the iodoform will be absorbed too slowly from these old cavities to be poisonous. The wound is then closed tightly, layer by layer, the deeper ones with catgut, and a surgical dressing applied. Good results will follow in the majority of cases.

Iodoform gauze is used more frequently now than any other medicated gauze. It is used for packing wounds about the natural orifices of the body because of iodoform's inhibitory action on bacteria. It is also very extensively used in packing tuberculous cavities after operation both in bone and in soft tissues. It should never be introduced into the abdominal cavity, as the peritoneum is very susceptible to the irritating and poisonous properties of iodoform. Iodoform gauze is rarely used as a dressing for fresh wounds, and its value in septic wounds is outweighed by its irritating properties and its disagreeable odor. It is believed by many to act as an inhibitory agent in suppurating wounds and is very commonly used as a drain for them, but it does not stop or control suppuration. Various strengths of iodoform gauze are prepared, but the ten-per-cent gauze will answer every purpose and is not so objectionable as a stronger preparation. Ten-per-cent gauze should contain, when dry, ten per cent by weight of iodoform. A ready method for preparing the gauze in small quantities is to rub the pulverized iodoform into sterile gauze which has been

slightly moistened. Manufacturers and large hospitals have their own formulæ for preparing it on a large scale. The following is a fair example of the various methods: "To prepare a ten-per-cent iodoform gauze take fifty parts by weight of gauze, forty parts of glycerin, and ten of iodoform. To properly incorporate the latter, the addition of two hundred parts of alcohol and one hundred parts of water is required. When this gauze is finished and dried the alcohol and water will have evaporated, while the glycerin and the iodoform remain, and the latter will then be ten per cent."

The Von Mosetig-Moorhoff bone plug is the iodoform preparation most recently introduced, and its use has certainly a very great advantage over any other method of treating bone cavities. After our experience with iodoform in other dressings we are naturally somewhat sceptical regarding its virtues in the bone plug, but the fact remains that the bone plug containing iodoform answers the purpose for which it was invented better than any other ever used, whether medicated or unmedicated. The correctness of the principle of filling a bone cavity with an artificial filling and closing the wound tight is established, but whether the efficacy of the filling is due to the iodoform in its composition remains to be demonstrated. This bone plug consists of sixty parts—by weight—of iodoform, forty parts spermaceti, and forty parts oleum sesami. These ingredients are slowly heated to 100° C. and, when allowed to cool, they form a soft mass which remains solid at the temperature of the body. For use it is heated to 50° C., being constantly stirred to keep the iodoform evenly distributed. This material does not act as a foreign body, nor as a culture medium. It possesses the inhibitory and medicinal properties of iodoform without causing iodoform intoxication. Its physical properties are such that it is gradually absorbed and replaced by granulations and finally by bone. Bone cavities are emptied of sequestra and detritus and are then sterilized by ninety-five-per-cent carbolic acid followed by alcohol; they are then thoroughly dried and filled with the melted preparation, which at the temperature of the body speedily solidifies. The periosteum and deeper soft parts are then closed with catgut, and the integument with silk or silkworm gut, and finally a surgical dressing is applied. The results in chronic osteomyelitis and tuberculosis are most gratifying. The method is not applicable in acute cases because the cavities cannot be made sterile.

LIGATURES AND SUTURES; SURGICAL DRESSINGS.

Sutures of various materials have been used by surgeons for the closure of wounds from a very early period. Ligatures for the control of hemorrhage were used by Celsus in the first century of the Christian era, and by Galen and others later, but hemorrhage was usually controlled by the cautery and hot oil until 1564, when Ambroise Paré earnestly advocated and practised the use of the ligature. It was not generally accepted, however, until about two hundred

years later, when Mr. Sharpe, a surgeon of Guy's Hospital, London, was largely instrumental in bringing it into general use.

Ligature and suture materials are of two varieties, the absorbable and non-absorbable. The principal varieties of the non-absorbable are silk, linen, silk-worm gut, horsehair, wire (silver, gold, iron), and Pagenstecher's celluloid hemp; the absorbable are catgut and kangaroo tendon. Non-absorbable ligature and suture material of some variety was the first in use, and was the best for closing superficial wounds until iodized catgut was introduced.

Silk has always been a favorite material for both ligatures and sutures. It is easily sterilized by steam or boiling, is smooth, strong, and ties securely. It is used by most surgeons at the present time for intestinal and integumentary sutures, and there are still many who use fine silk for ligatures and buried sutures. Many varieties of surgeons' silk are on the market, and most of them are good. The hard-twisted varieties are the best, and the black iron-dyed silk has the advantage of being easily seen. The fine and medium weights are all that are necessary. The finer it is the more certainly it can be made sterile and the less likely it is to act as a foreign body. In the days of septic surgery heavy braided silk was frequently used for ligating arteries, and the ends were left protruding from the wound so that it could be removed when it sloughed loose. At the present day it is not considered necessary to use heavy silk, and it is unwise to bury it, for obvious reasons. When heavy silk was used for ligating arteries, surgeons believed that it was necessary to tie the ligature tightly enough to cut through the inner coat of the blood-vessel, so that the latter would curl up and facilitate the formation of a clot. Since we have learned that only sufficient force is required to hold the walls of the vessel close together, fine silk, or preferably catgut, is used. When the heavy silk was used all wounds were septic and a large percentage of secondary hemorrhages occurred when the ligature came away. In those days the surgeon was anxious until the tenth or fifteenth day, when the ligature usually came away. The ligating of large arteries such as the carotid was an important undertaking in those days because secondary hemorrhage occurred in from fifteen to thirty per cent of cases and very frequently with a fatal result. The aseptic surgeon ties such an artery with a medium-sized catgut ligature with no thought of secondary hemorrhage, which is practically not considered in modern surgery. In the old days surgeons were afraid to tie veins because of the disastrous results from sepsis, but bleeding vessels of all kinds are tied now with safety and security. Without the aseptic ligature modern surgery would be impossible. Thorough hæmostasis is a very essential feature of our technique. In the septic era it was considered necessary to place the ligature, if possible, not less than one inch from any important branch for fear of hemorrhage, but now that is given secondary consideration. Senn has demonstrated by experiment that when an aseptic ligature is applied it is either removed by absorption or becomes encysted after

having performed the function of a provisional hæmostatic. He found that the lumen of the blood-vessel becomes obliterated by proliferation of new tissue from the endothelial and connective-tissue cells independently of thrombosis. The constricted portion of the vessel does not necrose, but becomes infiltrated with living tissue. After the application of a septic ligature the presence of an intravascular thrombus is a necessity, but after an aseptic ligature it is often entirely absent. When the intima is held in contact long enough the lumen is obliterated by the formation of a minute transverse scar strong enough to resist the blood pressure. As early as twenty-four hours after the application of an aseptic ligature the intima becomes adherent, and as early as after the lapse of three days Senn found a narrow, firm cicatrix underneath the ligature. Nature is throwing plastic material around the outside of the ligature at the same time, so that it is not necessary to apply a permanent ligature nor one that will last more than two or three days. In tying a silk ligature the first knot should consist of but a single reef, as this slips down snugly and will hold until a second knot is tied. Buried ligatures and sutures of silk can be used, but they are no better or safer than catgut and will frequently act as foreign bodies in spite of every precaution. One who is thoroughly familiar with the use of catgut would never think of using silk as a buried ligature, and will use it as a buried suture in intestinal work only. When the deeper parts of a wound are closed with catgut the integument may be closed with silk, but iodized catgut is better for this purpose. The various arrangements for storing and carrying sterilized silk so much employed a few years ago are little used now. It is better to sterilize the silk before each operation by steam or boiling. Steam is better because repeated boiling weakens the silk. After sterilization it should be kept in alcohol or sterile water during the operation. It should not be left exposed to the air. For carrying in an emergency bag it can be sterilized in the usual manner and then kept in bottles filled with alcohol or carbolic solution.

Horsehair is a favorite material for integumentary sutures with some surgeons. It should be prepared for use by first washing it thoroughly with soap and water and then it should be steamed or boiled. It should be kept in sterile water or alcohol during the operation.

Silkworm gut is the thread drawn from a silkworm killed when ready to spin the cocoon. It is used very extensively in surgery for stay sutures in deep wounds and for closing superficial sutures, and is a very valuable material for these purposes. It is pearly white, smooth, easily sterilized by steam or boiling, and does not furnish food for bacteria. It is kept in bundles of two varieties, the cheaper variety of which is composed of strands of various weights, while the more expensive variety is made of select heavy-weight strands. For general work the cheaper variety is very convenient, but for closing the abdominal wall the heavier variety only should be used, as the lighter strands are liable to break. This material can be sterilized by boiling with the instruments and

kept in sterile water during the operation. The boiling softens it sufficiently to make it tie well, and when once tied it does not slip. Some surgeons make but one double reef knot when using it, but an additional knot requires very little time and is safer. Silkworm gut, as a material for buried sutures has been found unsuitable. It does not undergo absorption and sooner or later acts as a foreign body. It is most commonly used for deep through-and-through or stay sutures in abdominal wounds, but the integument should be approximated by silk or iodized catgut, because the silkworm gut is too stiff to make a smooth running stitch. In some hospitals the silkworm gut is stained red or blue, to denote its presence under all circumstances. If properly divided it is easily removed after the healing of the wound. Stitch abscesses are comparatively infrequent from its use.

Pagenstecher's celluloid yarn is being extensively used as a substitute for silk in intestinal work and for closing superficial wounds. It is prepared by boiling linen thread for thirty minutes in a one-per-cent solution of sodium carbonate, washing in boiling water, drying between sterile towels, and finally soaking in a celluloid solution. This yarn is sterilized by steaming or boiling, is very firm and smooth, and does not absorb secretions readily. It is stronger than silk, and therefore a finer thread can be used. It is rigid enough to be readily threaded into the eye of the needle when wet, which is another great advantage over silk. It ties nicely, and when once tied is perfectly secure. It can be bought from instrument dealers in various sizes and is cheaper than silk. Linen thread was an old-time favorite with surgeons, but has been practically abandoned because of its unevenness and roughness, but the celluloid process overcomes these and other objections, so that it is doubtless destined to become an acceptable agent.

Silver wire still occupies a place in surgical technique, but not nearly so prominent a one as formerly, because in many places where it was once used the absorbable suture is now employed. In the early days of gynecology it was much used as a suture material by Marion Sims and others. Sims' early victories in closing vesico-vaginal fistula were doubtless largely due to the fact that he used wire sutures which were more likely to be sterile than other materials without thorough preparation. With asepsis, however, either absorbable sutures or silk may be used. There is at present but little excuse for using wire as a buried suture in the soft tissues, because the absorbable suture meets all the requirements and is free from the objections to wire. Wire can be made sterile by boiling, but, when left in the soft tissues, it too often makes trouble later by causing abscesses and fistulæ. It was applied quite extensively at one time in the operation for the radical cure of hernia. The silver filigree, a coarse mesh of silver wire, has been recommended as a filling in extensive ventral hernias, but its superiority over other methods has not been demonstrated. The application of silver wire in surgery is practically restricted to the treatment of fractures at the

present time, and even there the chromicized catgut is often preferable. In the treatment of fractured patella, for example, it has been demonstrated that the fragments can be held together by suturing the periosteum and surrounding fascia with chromicized catgut, thus avoiding the necessity for drilling the fragments, and the dangers of a foreign body in the tissues. There are many cases of fracture, however, in which silver wire is doubtless the best material at our command for holding the fragments together. It is strong, pliable, and can be made absolutely sterile by boiling. If the wound is aseptic it can, as a rule, be buried in these cases with no unpleasant after-effects. Occasionally, however, it will act as a foreign body some time later. In septic cases of compound fracture it is sometimes used with the expectation that it shall be removed later, but its use is not free from danger in these cases and its advantages are questionable. In wiring fractures the wire is passed through holes drilled in the fragments for the purpose, the fragments are adjusted, and the two ends of the wire twisted until the fragments are held snugly in place. It must not be twisted too tight or it will break, but it must be tight enough to prevent too free movement of the fragments. The end of the twisted wire is cut off half an inch or so from the bone and bent down flat against the bone so that it cannot interfere with the neighboring muscles. Silver wire has been recommended as a longitudinal suture for closing an abdominal wound, but it possesses a doubtful advantage over the absorbable suture usually employed, is more expensive and more difficult to apply properly. Wire should be prepared before each operation by boiling with the instruments, and should be kept in sterile water until used. The medium weight is best for ordinary use.

Absorbable sutures are, next to asepsis itself, the most prominent feature of aseptic surgery. Catgut and kangaroo tendon are about the only animal sutures now used, and the former is cheaper and more easily prepared, and meets every indication for ligature and buried suture so admirably that rarely is any other needed. Animal ligature was first suggested by Dr. Physic, of Philadelphia, in 1814. Dr. Jameson, of Baltimore, used deerskin cut into narrow strips and firmly rolled, and he tied all of the accessible arteries with it. Sir Astley Cooper tied the femoral artery with catgut. Nussbaum said, " Catgut is without doubt Lister's greatest discovery."

Catgut as prepared now is a universal favorite among those who have learned to use it. It did not gain its present high place in the estimation of surgeons without great opposition. Lister's methods of sterilization, as well as those of many others, were at times disappointing, and the plan of burying the catgut in the wound and leaving it there naturally became the target for severe criticism. It was the scapegoat for every slip in technique. It was customary for the surgeon who used a faulty technique and was constantly getting infected wounds, to blame the catgut instead of blaming himself, but the results obtained with it now silence criticism. There are a few good surgeons who do

not use it. The statements that it was not always sterile in former days, and that it may not be sterile now, are just as true of catgut as they are of silk or any other material, but it is equally true that it can be and is made sterile. The old objection that it does not last long in the tissues is now acknowledged to be a great virtue. The objection that it cannot be tied properly is disproved by every one who uses it with care. Secondary hemorrhage occurred when silk and other like materials were used, not because of the materials, but on account of sepsis. Surgeons who habitually use catgut rarely have secondary hemorrhages. Catgut swells after being in the tissues for a time, but the swelling has a tendency to tighten rather than loosen the ligature. In tying catgut it is necessary to make a double reef for the first knot, because one reef will not hold securely until the next one is made. The material is so slippery that the double reef may easily be tied, and it will hold securely while one or two additional reefs are tied. The facts that catgut can be broken when tied too tightly, and that it will slip when improperly tied, are commendable virtues, because they discourage tight ligatures and careless tying. It is not an uncommon error among surgeons, especially beginners in the use of catgut, to use too large a size. One accustomed to the use of catgut feels perfectly secure in using the medium and finer grades because his experience teaches him that they meet every indication. The heavier grades, aside from containing too much material, are more difficult to tie and to sterilize. When there is a doubt in the surgeon's mind as to the catgut having sufficient textile strength in a given case it is wise to use a double strand of a finer gut than to use a coarse strand, because the double strand affords the desired strength, exposes a larger surface, and is more readily absorbed. Experience has taught us that all kinds of ligature material will become encysted if aseptic, but that an inabsorbable material when tied around an artery may weaken the vessel at that place, while an absorbable ligature acts equally well as a temporary hæmostatic and is gradually replaced by living tissue, thus strengthening the vessel. Catgut can be hardened—so that it will last ten, twenty, thirty, or forty days in the tissues—by adding different percentages of chromic acid to it, making the chromicized catgut of commerce. The principal advantage of the chromicized gut is that it enables the surgeon to use a finer strand. The heavy chromicized gut and that containing an excessive amount of the hardening agent are objectionable because the knots undergo absorption very slowly and are liable to act as a foreign body. For this reason the chromicized gut should not be used when the plain gut will answer the purpose just as well. Infection occurs more frequently with the chromicized than with plain catgut. The use of the chromicized gut is largely a habit; it is rarely necessary, because the plain gut meets most requirements. Plain catgut is not a good material for superficial sutures which are required to carry any weight, because the lighter-weight gut becomes absorbed too quickly and the heavier readily becomes infected from outside or from the bacillus which

has its habitat in the skin. The iodized catgut of medium weight is, however, admirably adapted for this purpose because it lasts long enough, does not become infected, and does not have to be removed.

Buried sutures are a most essential feature of aseptic surgery, and the best material for the purpose has long been a moot point among surgeons. The essential requirements of an ideal material for buried sutures are, first, absolute sterility; second, adequate and uniform tensile strength; third, flexibility, so that it will tie easily and securely; and, fourth, a proper degree of absorbability. Catgut as now prepared meets with all these requirements perfectly, and no other material does. Some surgeons maintain that an ideal catgut must be mildly antiseptic. They claim that the presence of the antiseptic prevents the softened catgut from acting as a culture medium; but the soft tissues are very intolerant of chemicals of all kinds and it is an open question whether the antiseptic does not do more harm than good, for we have long since learned that wounds heal better when no chemicals have been applied to them. Silk and other inabsorbable materials, including catgut with an overabundance of hardening material which makes it practically inabsorbable, have been extensively used for buried sutures, but they are objectionable and unnecessary. They are objectionable because they more often than catgut act as foreign bodies, causing abscesses and sinuses. They are unnecessary because they are useful only during the healing of the wound, which is accomplished in a few days, and common catgut meets this indication and then disappears, while the inabsorbable material remains and is likely to cause harm. If, after the healing of the wound, the newly formed tissues begin to give way, the sutures are powerless to hold them because pressure necrosis takes place and they cut through. It is well known that when for any reason a superficial wound fails to unite, the stitches not only fail to prevent the wound from gaping, but soon become a source of irritation and stitch abscesses and must be removed. When a post-operative hernia occurs after the use of an absorbable suture there is no suppuration or sinus further to weaken the parts; but when unabsorbable sutures have been used they cause pressure necrosis and may and often do cause abscesses and sinuses which not only aggravate the hernia but cause the patient pain and expose to danger.

Kangaroo tendon has been introduced and ably advocated by Dr. Marcy as a substitute for catgut as a suitable material for buried sutures. It is a good material, but catgut is better, cheaper, and easier to prepare. The larger size of the kangaroo strand, once claimed as an advantage, is now believed by many to be a disadvantage.

That there are so many *methods of preparing catgut* is evidence that its preparation is important and by no means simple. It requires much more care and trouble for its sterilization than the various inabsorbable suture materials, but its superiority over all those materials makes it well worth while.

That catgut can be perfectly sterilized by several methods is now an established fact, but no one method is yet generally accepted as the best. Unfortunately, laboratory tests after chemical preparation of catgut are not reliable because methods of removing the chemical from the catgut before the bacteriologic test are not eminently practical. Catgut cannot be sterilized by boiling in water or by steaming because it is thus destroyed, but it can be boiled in other media and be sterilized by dry heat. Chemical methods were at first quite disappointing, but some of them have become so perfected that they are now quite reliable. The so-called catgut is prepared from the fibrous coat of the small intestine of the sheep. The best catgut on the market comes from the German manufacturers and the exact method of its preparation is somewhat of a trade secret, but it is evidently prepared with greater care than formerly because it is frequently sterile without special preparation. Different manufacturers prepare different sizes of catgut, designating the different sizes by number. Unfortunately, each manufacturer has a scale of his own which may lead to mistakes when ordering the material. Three sizes—fine, medium, and coarse—are sufficient for all purposes. The medium size is the one most commonly used. The finest strand is used only where it is necessary for it to hold for one or two days, or when it is employed upon small arteries and the peritoneum. The medium size will last five or six days in the tissues, which makes it suitable for ligating arteries, excepting the very largest, and for buried sutures generally. The heavier strands are seldom used, surgeons generally preferring to use chromicized gut when a more durable ligature or suture material is required. The medium-weight catgut has all the tensile strength ever required. The day of tight ligatures is past, and one who breaks the medium weight catgut is using more strength than is necessary.

Claudius' method or *the iodine method* is one of the most recent and popular chemical methods of catgut sterilization. Senn has used this method exclusively for over two years with entire satisfaction in his clinics and in private practice. During this time he has had hundreds of cultures made without positive results in a single instance. The method is thus described:

"The commercial raw catgut, without any preliminary preparation, is wound on a glass roll, two strings tied together to each roll. The sterilization is effected by immersion in a one-per-cent solution of iodine. The solution is made by dissolving one part of iodine and one part of potassium iodide in one hundred parts of water. The potassium iodide is dissolved in a small quantity of water to which the iodine, finely pulverized, is added and the concentrated solution is then diluted to one per cent. The solution and catgut are kept in a bottle with a wide mouth which is closed with an accurately fitting glass stopper. The date is written on the label of the bottle. After eight days the catgut is ready for use, and is preserved in the same bottle and in the same solution.

Before being used, the catgut is immersed on a glass roll in a three-per-cent solution of carbolic acid, or an indifferent sterile fluid, for the purpose of removing the iodine from the surface of the threads. The threads are cut in the solution as they are needed. The catgut not used in the operation is returned into the bottle, thus doing away with unnecessary waste of material. The catgut thus prepared is pitch black, soft, pliable, and almost as strong as silk. The knots are firm and not liable to slip or become untied. Catgut prepared by this method, according to its size, resists absorption for from twelve to sixteen days. Iodized catgut is not only absolutely sterile, but at the same time is antiseptic. The catgut remains in the aqueous iodine solution, without loss of tensile strength, for several weeks. If it is desirable to preserve it for a longer time it is advisable to transfer it after eight days to an alcoholic solution of similar strength." It cannot be kept long in sterile water because the water removes the iodine and softens and destroys the gut. This is certainly the simplest of all methods of catgut sterilization and the one that naturally appeals to those who prepare their own material. Some who gave the iodine catgut a trial have abandoned it because it soon became brittle, and others because the iodine was too irritating. Senn claims that the brittleness is due to the material used and not to the method.

Abbott, of Minneapolis, has found that, by using but half the quantity of iodine recommended by Claudius, the catgut is quickly and surely sterilized and does not become brittle or irritating. He uses but two sizes, the fine and the medium. He finds that the fine thread prepared in this manner lasts one week and makes a very satisfactory cutaneous stitch. He prepares the chromicized gut by immersing the raw catgut for twenty-four hours in a 1 : 2000 aqueous solution of chromic acid, after which it is sterilized in the iodine solution in the usual manner. After eight days he transfers the iodized gut to ninety-five-per-cent alcohol and uses it from that medium. To avoid the tendency to become brittle he suggests that it is better to prepare the gut frequently in smaller quantities than to prepare a large quantity which must be kept in solutions for a long time. He has used catgut prepared in this manner for two years and is perfectly satisfied with it. He believes that this method is superior to all others because it is so simple and so efficient.

We are greatly indebted to Moschcowitz, of New York, for much valuable information concerning iodine catgut. He and Gerster have used it in Mount Sinai Hospital for nearly three years and in no case have they had the slightest occasion to regret their confidence in it. Moschcowitz, like many others, found that after a time catgut prepared and preserved by the original Claudius method had a tendency to become brittle, and, like Abbott, he has been experimenting to find and remove the cause. He has learned that when the gut is removed from the iodine solution at the end of eight days and kept in a dry sterile jar it does not become brittle, and he has been using this dry gut for the past nine

months with perfect satisfaction. "It is used dry, just as it is cut from the spool, without any previous immersion in carbolic solution or sterile water. Any unused catgut may be resterilized on a future occasion." He says that this dry gut is somewhat stiff like a fine wire, but that it has no tendency to kink up, and when used it soon becomes soft. He knows of no disadvantages connected with this gut. He has made a large number of experiments, from which and from his practical experience he draws the following conclusions:

1. The "dry" iodine catgut is absolutely sterile.
2. It is impossible to infect it by ordinary means.
3. Its imbibition with iodine is not sufficient to make it act as an irritant upon the tissues.
4. Its tensile strength is superior to that of raw catgut and to that prepared by the sublimate-alcohol method.
5. It is easily and cheaply prepared.
6. It is absorbed only after it has served the purpose for which it was intended.

The Bartlett Method.—This method, devised by Dr. Willard Bartlett, seems destined to supersede all other methods of preparing catgut. The sterilization is effected by the use of both heat and iodine. The boiling in petrolatum destroys every vestige of germ life and at the same time renders the gut soft and pliable. The subsequent preservation in an iodine solution renders it antiseptic and protects it from infection; at the same time the iodine does not seem to irritate the tissues as other chemicals do. The following is Dr. Bartlett's description of his method:

"The process can be divided into three steps: (1) The physical preparation of the material; (2) its sterilization; (3) its storage.

"1. The ordinary commercial ten-foot catgut strand is divided into four equal lengths, each of which is made into a little coil about one inch and a half in diameter. By twisting the last free end about four times around this little coil the latter will maintain its shape. These coils in any desired number (I usually take about one hundred and twenty of them at a time) are strung on a thread, like beads on a string, in order that the whole number may be handled at once. This string of coils is hung in a metal can, better still in a beaker glass, but is not allowed to touch the bottom or sides. I suspend them by carrying the two ends of thread through a small opening in a pasteboard cover which is placed on the receptacle. The same opening serves to admit a thermometer, which is carried down to exactly the point where its mercury bulb is on a level with the topmost coils. Liquid petrolatum is now poured in, the quantity being sufficient to immerse the catgut and the bulb of the thermometer.

"2. The vessel is set upon a pan of sand under which is placed a tiny gas flame of merely sufficient intensity to raise the temperature of the oil to 212° F. within from one to two hours. A little practice enables one to guess the size of flame

necessary for this purpose. This is best done in the evening, and the temperature should be allowed to remain at about 212° F. (a variation of a few degrees does not matter) until morning. The heat is then increased to such an extent that the temperature will run up to 300° F. in the course of an hour; then the gas is turned off and the temperature of the oil allowed to return to about 212°.

"3. The pasteboard cover, together with the string of catgut coils, is lifted off, the superfluous oil is allowed to drop off, and then the thread is cut, permitting the coils to drop into the following mixture:

Columbian spirits	100 parts
Iodine flakes	1 part

"The catgut is now ready for immediate use, and will keep without deteriorating for any length of time. The jar may be opened any number of times so long as a sterile instrument is used for removing the coils, since the iodine protects the coils that are left behind from accidental contamination."

In a personal communication from Dr. Bartlett in reply to an inquiry concerning his method of chromicizing catgut, he makes the following statement:

"I have given up the use of chromic or other acids in the treatment of catgut, and use formalin vapor where I desire to make catgut last longer than usual. I simply suspend the coils a few inches above a ten-per-cent formalin solution in an airtight vessel for twenty-four hours. This is far simpler than immersing it in the solution, and the catgut requires no washing afterward. No. 2 catgut so treated will last for between two and three weeks in muscle tissue."

Ochsner's Method.—The following method of preparing catgut has been employed at the Augustina Hospital for years with perfect satisfaction. "Catgut is prepared by immersion in sulphuric ether for one month, then for one month in strong commercial alcohol in which one grain to the ounce of corrosive sublimate has been dissolved, the solution being renewed once during this time. It is then preserved indefinitely in a solution of one part iodoform, five parts of ether, and fourteen parts of strong commercial alcohol. It should never be handled by any one except the surgeon and the chief assistant. This catgut will last seven to ten days in tissues, according to the size used. It is employed in all ligatures, both in the peritoneal cavity and elsewhere, and for all buried sutures except in hernias and in the suturing of bones. For these purposes a chromicized catgut is employed, which lasts for from fifteen to thirty days according to size. This is prepared after the following formula: The catgut is immersed in ether for one month, then in a solution prepared in the following manner:

℞ (A) Chromic acid	1 part	(B) Take of solution A	1 part
Water	5 parts	Glycerin	5 parts
(Carefully dissolve.)			

Take solution B and soak therein catgut for forty-eight to ninety-six hours, according to the resistance wanted. That soaked for forty-eight hours will resist absorption by the tissues for fifteen days; that for ninety-six hours will resist for thirty days. (C) Take catgut out of solution B, rinse quickly in sterilized water to free it from solution B, stretch and rub quickly with a hard, sterile towel to remove any of solution B which may still be adhering to it, wind on rods or slides at least three inches in length, and preserve indefinitely in the following solution:

(D)	Carbolic acid 95 per cent	1 part
	Glycerin	5 parts

The catgut may remain in this solution for many months without depreciating in quality, or it may be kept for an indefinite period of time in the same solution as the ordinary catgut, composed of:

Iodoform	1 part
Ether	5 parts
Strong alcohol	14 parts

The jar containing the ether in which the catgut is kept for one month should be filled only about one half with the loose coils of ligature and then it should be filled with ether; it should be closed airtight and should be picked up every day or two and shaken in an inverted position in order to wash off any substance which may accumulate upon the surface of the coils. At the end of two weeks the ether should be removed and fresh ether substituted. The same precaution should be taken with the solution of corrosive sublimate in alcohol. It is especially important not to wind the catgut tightly before placing it in these solutions, because this may prevent the solutions from penetrating all parts of the material. One precaution is necessary in the use of catgut prepared in this manner—it must not be placed in water before it is used at the time of the operation."

Koenig's method of *boiling catgut in cumol* is a good and reliable one, but is only suitable for large hospitals where they have a special apparatus for this purpose. This method as modified by Clark and Miller is used in St. Luke's Hospital, of New York, and in Johns Hopkins Hospital, of Baltimore, with perfect satisfaction.

The New York Hospital Method.—"The raw gut is put in benzin for twenty-four hours to remove fat; it is then wiped dry, wound on glass spools, and boiled in alcohol for an hour to an hour and a half, the time varying according to the size of the gut. After twenty-four hours the gut (still remaining in the alcohol) is boiled for half an hour to kill any spores which may remain, and is then ready for use. It is kept in sterile alcohol until used." This gut is sterile, but breaks more easily than that prepared by the cumol method.

The Boeckmann method of sterilization by dry heat is a most excellent one but requires special apparatus and extra care in its management, so that it is not

a choice method except for manufacturers. In the Northwestern Hospital, of Minneapolis, and in many other hospitals in the Middle West, the Boeckmann catgut, prepared in the Ramsey County Medical Society's laboratory, has been used for years with great satisfaction. This gut can be bought plain or impregnated with pyoktanin. It is sterile, and when dipped in sterile water just a moment before using ties very easily and securely. The best feature of this gut, aside from its reliability, is that it is put up in small paper envelopes which can be carried more conveniently than bottles or glass tubes. It is wrapped in two layers of paraffin paper and then hermetically sealed in a small paper envelope and properly labelled. This is all done before the sterilization, thus avoiding all handling of the gut afterward. These envelopes are placed in a hot-air sterilizer and kept at 280° F. for three hours or more, which makes the gut perfectly sterile. The envelope can be taken in unsterile hands, the end torn off and the enclosed coil of gut with its paraffin wrappers dropped into a sterile hand.

The writer has used catgut prepared by the Boeckmann method for a number of years with great satisfaction, but he is now introducing the Bartlett catgut into the Northwestern Hospital because it is much cheaper and more suitable for superficial stitches.

Cargile membrane is a sterile animal membrane first prepared for the prevention of peritoneal adhesions and later adapted to a variety of purposes in wound treatment. It is prepared plain and chromicized and compares in absorbability with plain and chromicized catgut. Experimental and practical use demonstrate that it is of limited advantage in preventing adhesions in some instances, but that its use in the peritoneal cavity is not all that had been hoped for, because it acts as a foreign body and becomes covered over by omental adhesions. Morris, Deaver, and others have used it in the human abdomen, but as yet there has not been sufficient opportunity to examine the results and thus to establish its real value. Craig and Ellis conclude that the chromicized membrane is of decided help in preventing the formation of adhesions to wounded nerves and tendons when surrounded by contused tissues. They recommend that three or four layers of the membrane be wrapped around the nerve or tendon. They suggest that the chromicized membrane may be valuable as a temporary dura in brain injuries, but its value for this purpose has not yet been demonstrated. It is made from the peritoneum of the ox and prepared in the same manner as catgut. It can be bought from reliable manufacturers in any desired quantity.

Adhesive plaster is a very valuable aid to the surgeon in many ways and, as now made by responsible manufacturers, is clean and trustworthy. The old-time diachylon plaster, which required heat to make it adhesive, has been almost entirely superseded by rubber adhesive plaster which is adhesive at all temperatures. The ordinary rubber plaster is usually bland and unirritating and

perfectly adapted to surgical uses over a surgical dressing or where the skin is unbroken, but it is not aseptic and is therefore unfit for application directly to a wound. Adhesive plaster prepared at the suggestion of Dr. Lilienthal, of New York, is aseptic and especially adapted to the closure of wounds in certain locations. The ordinary plaster contains impurities and is destroyed by efforts at sterilization; but in the manufacture of the aseptic plaster the impurities are removed. It is cut into strips of suitable widths, enclosed in double envelopes, and sealed to prevent infection. This plaster has the great additional advantage of being unaffected by moisture. Adhesive plaster exposed to the air soon deteriorates, but when kept rolled as it is found in the shops it will keep for a reasonable length of time. Before the plaster is applied the skin should be shaved, freed from grease, and made perfectly dry. When it is applied to an extremity it should be remembered that it may restrict the circulation just as a tight bandage would. Whenever practicable an adhesive-plaster dressing should be covered by a gauze roller bandage which keeps it clean and prevents it from adhering to clothing or bedding. A quick removal of adhesive plaster will give the patient the least annoyance. If it is adherent to hair a little ether will facilitate its removal.

The uses to which adhesive plaster are put in modern surgery are too numerous to mention, but it may not be amiss to call attention to a few of the most important ones. In the treatment of fractures it is indispensable in the application of traction. For fractures of the femur the plaster spread on heavy cloth called moleskin should be used because it is strong enough to last throughout the treatment, thus saving the patient the discomfort and danger of a change before the bone is united. Sayre's dressing for fractured clavicle, Moore's dressing for Colles' fracture, the dressing for fractured ribs and fractured patella with adhesive plaster, are among the best of their class. Gibney's adhesive-plaster dressing for a sprained ankle enables a patient to get about much sooner than when the condition is treated by the older methods. In varicose ulcers of the leg adhesive strapping is especially beneficial. The dressings of a stump after amputation may be held in place by strips of plaster extending well up on the integument above the dressing. Dressings applied to any part of the body can be held in place by adhesive strips.

For an abdominal wound the adhesive strips answer a double purpose when properly applied over the dressings. They prevent the dressings from slipping and give support to the wound, thus relieving tension upon the sutures. The use of adhesive strips has much to do with preventing stitch abscesses and post-operative hernias in present-day surgery. To apply them successfully requires that they should be so adjusted that they give support to a wound without constricting it. For abdominal work the strips should be about one inch and a half wide, and should begin well around on the patient's back on either side and extend a little beyond the wound so that the ends shall overlap at the

wound. At the end next the wound each strip should be folded upon itself with adhesive surfaces together so that, for a distance of three or four inches, the strip shall present no adhesive surface. These ends can be overlapped and fastened together by safety pins. They can be tightened or loosened at will, and they permit a change of dressings without subjecting the patient to the discomfort of having the adhesive straps pulled from the skin.

Patients who have undergone an abdominal operation usually feel the need of some kind of abdominal support. When the wound is an aseptic one this need can be met by properly applied adhesive strips better than by an abdominal binder which is bulky and which will not readily stay in place. Patients suffering from prolapse of a kidney or some other abdominal organ can often be relieved by adhesive strips so applied as to render a surgical operation unnecessary. The improvement in neurasthenic patients with distended abdomens and ptosis of all the abdominal organs following support of the parts by adhesive strips is sometimes remarkable.

Adhesive plaster was used for the closure of wounds years ago, but in those days all methods of wound closure were less promptly effective than they are to-day, because the wounds were septic. In early antiseptic days this method could not be satisfactorily employed because the dressings were moist; but, since the advent of aseptic surgery with dry dressings and since aseptic adhesive plaster is obtainable, it is quite possible to close certain wounds in this manner. Without the buried suture, plaster would rarely meet the requirements for wound closure because it would not obliterate dead space and it would have a tendency to roll the edges in; but the employment of buried catgut sutures in properly adjusting all the deeper tissues and obliterating dead space has rendered it possible to close the integumentary wound by aseptic adhesive plaster, and this plan is used almost to the exclusion of superficial stitches by some surgeons. This method of wound closure is only applicable when the wound is perfectly dry and when it closes without tension. Under proper conditions it insures healing with the minimum amount of scar. It can be applied in much less time than sutures and can be removed easily and quickly. Nervous patients usually have an unwarranted amount of anxiety about the removal of the stitches, and if they can be assured that there are none to remove they are saved much worry. Adhesive plaster is an indispensable part of an orthopedist's armamentarium. He uses it extensively for adjusting apparatus to the body and to protect the integument from chafing. It affords a very efficient means of pressure upon an inflamed joint.

Plaster of Paris in the form of plaster bandages is a valuable surgical appliance. For ordinary use good bandages can be bought already prepared, but when large quantities are used, as in a hospital, it is much more economical to prepare them. The prepared bandages are put up in airtight tin boxes to protect them from moisture. They vary in width from two to four

inches. A three-inch bandage is a good width for general use. In preparing bandages coarse crinoline cloth and the best dental plaster only should be employed. Cheese cloth is too heavy and too closely woven, and commercial plaster is too slow-setting. All starch and glue should be removed from the crinoline because it interferes with the setting of the plaster. The bandages must be rolled loosely and should have the meshes of the cloth rubbed full of the plaster as they are rolled. Either an excess or a deficiency of plaster makes a poor bandage. The bandages must not be moistened until everything is ready for their application. They should then be stood on end in a large basin well filled with warm water, one at a time. A small amount of water soon becomes so saturated with plaster that it will not soak through the bandages. Cold water makes the plaster harden or "set" too slowly, and hot water has the opposite effect. When the air bubbles cease to arise from the bandage it is ready to be taken out of the water. It should then be removed promptly, gently squeezed in the hand to eliminate the excess of water and applied as a roller bandage is applied, save that reverses need not be made. Each layer is thoroughly rubbed with a moist hand as it is applied so as to make it coalesce with the layer underneath. The rubbing also takes the place of reverses and makes a smooth surface. As one bandage is taken from the water another should be put in. If several are put in at once some are liable to set before the surgeon has had time to apply them. Objections have been made to plaster on the grounds that it is too heavy, becomes filthy, and is difficult to remove. These objections can be applied to any material when too much of it is used or when it is improperly applied or when it is long retained in place. The most common mistake made in the use of plaster of Paris is that too much is used at one time, the dressing thus being rendered too heavy and difficult to remove. When good bandages are properly applied to an extremity the splint when removed should not weigh much more than a splint of heavy pasteboard or light sole leather. A heavy plaster bandage is unnecessary, is a burden to the patient and a waste of material, and is more difficult to remove than a lighter one of the same material. The removal of a plaster bandage by unskilled hands is difficult, but when properly done it is an easy matter. The

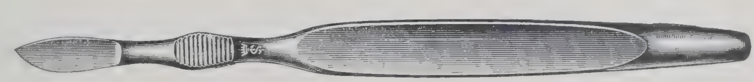


FIG. 282.—Plaster-Bandage Cutter.

plaster-cutters on the market are not satisfactory and are quite unnecessary. An ordinary pocket knife with a good strong blade will answer the purpose admirably, but a knife with a short stout blade and a solid metal handle ending in a wedge with which the edges of the splint can be pried apart is the best instrument for the purpose (Fig. 282). To remove a plaster splint a strip

about an inch wide at the proper place should be moistened. This makes the plaster soft so that it will cut easily. No solution is better for this purpose than water. After they have been moistened, the fingers of the left hand should grasp the upper end of the splint and lift it far enough from the part for the knife to be applied to its upper edge. One side of the cut splint should be constantly pulled away from the skin so that the knife can be applied to the edge just as a shoemaker cuts sole leather. When the procedure is carried out in this manner a properly applied plaster splint can be removed from the whole length of the lower extremity in a brief time, but, when the knife is applied to the outer surface of the splint instead of the edge, the undertaking is difficult and tedious.

Plaster is used very extensively in the treatment of tuberculous joints to secure rest and is considered the best material at our disposal by many surgeons. For this purpose it must extend well above and below the diseased joint in order to limit motion as much as possible. For the treatment of clubfoot, plaster is undoubtedly the best dressing in ordinary hands. The plaster should not be used to overcome the deformity, but to maintain the correct position after the deformity has been reduced. If an effort is made to overcome the deformity by force after the plaster is applied, pressure necrosis will surely follow. When necrosis of the integument follows the application of the plaster bandage the fault is not with the material. Pott's disease at the lower part of the spine can be satisfactorily treated by the plaster jacket, and, even in the hands of one accustomed to applying braces, is more likely to be efficiently applied than a steel brace.

For the treatment of fractures, plaster, properly applied, is one of the very best retaining materials. It is easy to obtain and to apply, and remains where it is put notwithstanding meddlesome patients and friends. It is not a suitable material where much contusion is present and much swelling is to be expected. It should not be used as the first dressing in any case where the surgeon or his assistant is not near the patient, for fear of swelling, but after a week or ten days it can be made a satisfactory dressing for most fractures. For the treatment of compound fractures there are possibly better splints than plaster of Paris, but in the hands of those accustomed to its use it is one of the best materials for this purpose. It is applied over the surgical dressing, and a window cut to permit treatment of the wound.

Plaster bandages are often applied over a surgical dressing to keep it in place and to keep the parts at rest. For example, it is often advantageously applied after a hernia operation on a child.

Rubber goods are used very extensively in surgery at the present time. Rubber gloves have already been considered on page 709. Rubber tubes still hold an important place as drainage material, and, when a tube of any kind is indicated, this is probably the best. Tubes of a very small size are no better than strips of rubber tissue, and very large tubes are no longer used because they are unnec-

essary. Tubes varying in size from that of an ordinary lead pencil to that of the little finger will meet every indication. The coarse white rubber tubing of commerce should not be used except in an emergency. The soft red or black rubber tubing made purposely for drainage is the best. It should be soft and flexible, with walls thick enough to prevent collapsing, but not so thick as to make the calibre of the tube too small. It can be prepared by boiling and kept in a five-per-cent carbolic acid solution. It is safer to boil tubes just before the operation than to depend upon those which have been kept in a disinfecting fluid.

The Esmarch bandage, as originally introduced for emptying out the blood from an extremity preparatory to a bloodless operation, consisted of a rubber webbing, but this has given way to the solid rubber bandage known as Martin's bandage. Most of the Martin bandages found in the instrument stores are apt to be practically worthless for the purpose for which they are intended because they are of too light weight. They are too frail to force the blood out of a large extremity and they break easily. A good rubber bandage for this purpose should be as thick as blotting paper and three or four inches wide. It can be boiled, or prepared for use in carbolic solution.

Rubber tissue is used in surgery for drainage, to cover skin grafts, and to cover a damp surgical dressing and keep it moist. When very little drainage is required a small strip of rubber tissue answers the purpose admirably. It is very extensively used for surrounding gauze drains to prevent them from adhering to the tissues. After skin grafting the rubber tissue is cut into strips and laid over the grafts like the shingles on a roof. This permits the discharges to escape into the gauze dressings applied over them and prevents the gauze from adhering to and destroying the grafts. Rubber tissue cannot be boiled because the heat destroys it. It can be prepared by washing in soap and water, rinsing in clear water, and being placed in a 1 : 1,000 solution of bichloride. The water in which it is washed cannot be very warm or the tissue will adhere to itself and be destroyed. The bichloride solution should be changed at least once in two weeks and, just before using, the tissue should be immersed in a sterile salt solution to remove the bichloride. Rubber tissue cannot be kept satisfactorily in a carbolic solution because it is softened thereby, and becomes, when it is in this condition, adhesive and inconvenient to handle.

INSTRUMENTS.

A young practitioner just purchasing his first supply of instruments will find it more economical and in every way more satisfactory to buy only such as are necessary to meet his requirements, and then later to add to this stock as his needs arise. Always buy the very best quality, since they are more economical in the end and serve their purposes better.

Instruments should be smooth in finish and should be made of metal throughout, only the best of material being used. The beautiful pearl, ivory, ebony, tortoise-shell, and wooden handles so much in vogue at one time are no longer used because they readily become infected and cannot be sterilized without injury. Nickel-plating keeps instruments from rusting, but should never be applied to cutting instruments. An instrument should never be made heavier than is necessary to give it the requisite strength, for light instruments are conducive to delicate work. Too long an instrument is a mechanical disadvantage as it keeps the operator's hand too far away from the field of operation. The nearer the hand is to the wound the quicker and more accurate the movements. Most scissors, hæmostatic forceps and clamps are now so constructed that they can be readily taken apart when being cleaned, but a screw lock is much better for scissors and hæmostats because it can be tightened as it becomes

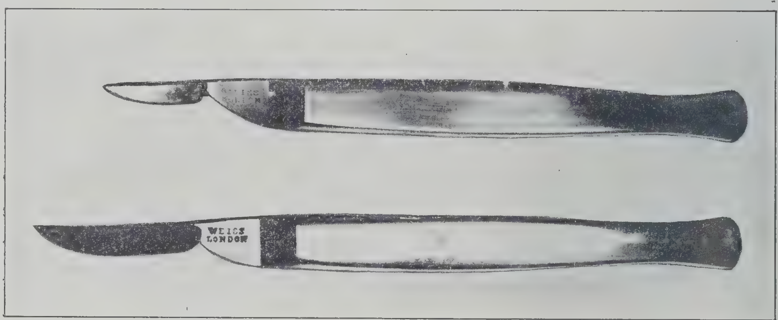


FIG. 283.—A Very Good Pattern of Knife; small blade and smooth metal handle.

worn. Simplicity in construction is a commendable feature in an instrument. Complicated instruments made for special operations are to be avoided when the work can be properly done with ordinary instruments. It is too much the tendency among instrument makers and young surgeons to invent new instruments or change old ones. One who allows himself to purchase the wonderful things presented by the travelling instrument dealer will soon have his instrument case filled with a lot of worthless implements. There are a few special instruments that are almost indispensable, but as compared with the number offered for sale they are very few indeed. It is a mistake to have too many instruments prepared for an operation, as comparatively few are ever used, and the rest are only in the way and are deteriorated by boiling. Every surgeon has his favorite instruments with which he becomes specially dexterous. The good surgeon depends upon his experience and skilled hands to enable him to do good work with whatever instruments may be present in an emergency. Instruments should be kept in good repair, because a dull cutting instrument bruises the tissues and makes the surgeon's efforts slow and uncertain, and a forceps that does not bite true or lets go is always

disturbing and sometimes dangerous. It is bad taste to make an unnecessary display of instruments. Bright, clean-looking instruments will surely make a better impression than black, dirty-looking ones. The surgeon or the institution using instruments until they become rusty or black should have duplicates, so that one set can be repaired or polished while the other is in use. This is really a matter of economy, for an instrument properly cared for will last much longer than one that is neglected.

Knives.—A good sharp knife is very essential for quick, smooth operating.

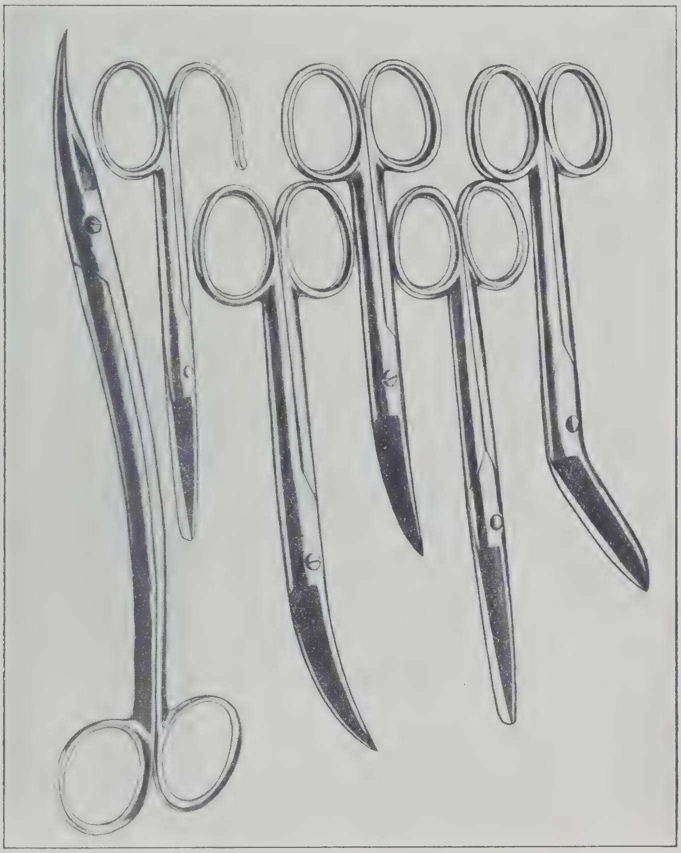


FIG. 284.—Various Patterns of Surgical Scissors. With this assortment all the conditions in general surgery can be met.

It should have a blade of the very best steel and a smooth metal handle of sufficient weight to balance well in the hand. The blade should be comparatively small and bellied or convex on the cutting edge. When it is too long it cannot be so skilfully handled, and length is never necessary except in larger amputating knives, because the cutting is all done by a portion of the blade near the point. The handle should be so shaped that it can be used as a blunt dissector when necessary (Fig. 283). Knives should always be cared for by some

one connected with the operating-room, so that they may always be in order. It is dangerous to use a dull knife, and then without warning employ a sharp one.

Scissors.—Next to knives, scissors are the most important cutting instruments. They too should be of the very best steel and should be kept sharp. The blades should be united by a screw pivot which should be kept tight enough to keep the edges in proper relation with each other (Fig. 284). Infection and consequent failure of union may be caused by necrotic tissue that owes its origin to the pinching of dull or loose-jointed scissors. Scissors should not be nickel-plated. Boiling dulls them and eventually spoils them. They are made in shapes and sizes too numerous to mention, but with a small assort-

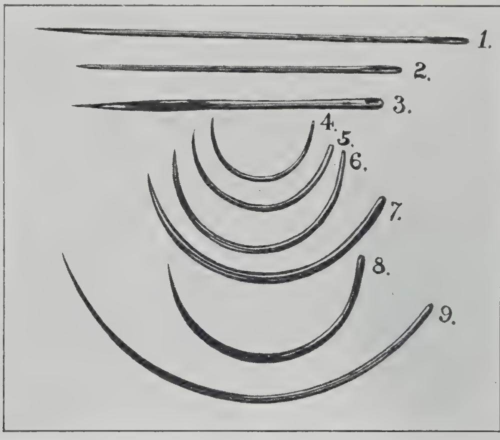


Fig. 285.—Various Kinds of Needles for Surgical Purposes. 1, Cutting point, for integument; 2, round point, for use in operations upon the intestine; 3, saddler's needle, for integument; 4, 5, 6, and 7, round-pointed, for intestine and peritoneum; 8 and 9, needles with a cutting edge, for integument, muscle, and mucous membrane. The curved needles are all flat on the concave and convex sides and have eyes large enough to take catgut suture thread.

ment of medium-sized straight and curved scissors most requirements can be met. It is usually in deep wounds with dense tissues that the large blunt-pointed varieties are needed.

Needles.—The half-curved cutting-edged needle so commonly used twenty-five years ago is rarely used now, the straight and full-curved needle having taken its place. The straight needle is always to be preferred when applicable, because it does not turn in the fingers or needle-holder and the operator knows where the point is located. For the integument, for intestinal, and for stomach work a straight needle is better, while for deep wounds and plastic work some form of curved needle is preferable. For intestinal sewing the point of the needle should be round like the ordinary cambric needle, but for the integument it should be spear-pointed or have cutting edges like the saddler's needle. The curved needle is usually made with too much cutting edge. If the

needle has a sharp point with a cutting edge extending half an inch from the point it will perforate the tissues readily and will not cut the operator's fingers. For peritoneal work the curved needle should have a round point without a cutting edge. Every curved needle, except the large ones intended for applying through-and-through sutures, should have an eye large enough to be threaded with catgut of a medium size. It should be flattened on both the concave and convex sides for some distance from the eye so that it can be held securely in the needle-holder. A curved needle that is round throughout its length except at the eye is unreliable and unsafe and should never be bought. It is unreliable because it is liable to turn in the holder at a critical moment, and unsafe because when it turns it may puncture an important part. It is also very liable to break because the operator is apt to grasp the eye in the

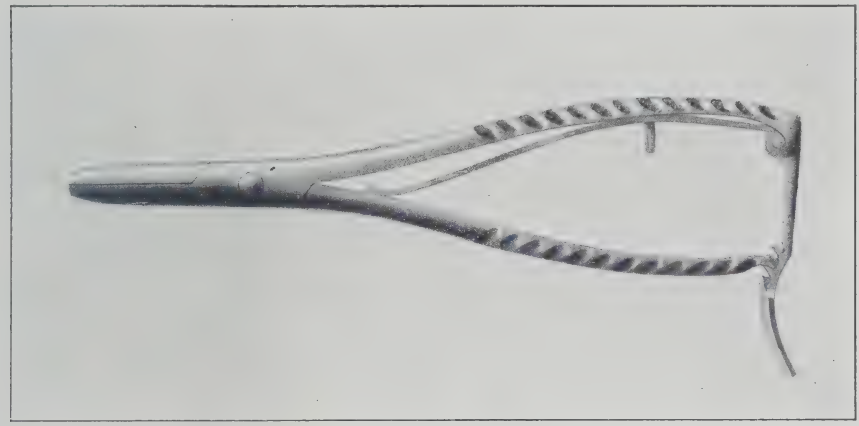


FIG. 286.—A Very Satisfactory Form of Needle-Holder.

holder and to apply greater pressure than the needle can withstand in his efforts to hold it securely (Fig. 285).

Needle-Holders.—A perfectly satisfactory needle-holder has not yet been made. This is proven by the fact that new ones are constantly being designed. The requirements of a needle-holder are: that it shall not be bulky and heavy; that its catch shall be simple in construction and easy to work; that it shall hold the needle firmly without breaking it; and that its jaws be so constructed that the point of a needle can be quickly grasped after it has been passed through the tissues so as to draw the needle and thread through. Every surgeon becomes accustomed to one of the many varieties, but he always expects a certain amount of annoyance and inconvenience from it and is usually ready to try another which appears to be an improvement. Many of the faults attributed to the needle-holder are really due to improperly shaped needles. A curved needle which is round throughout its length cannot be properly held by a flat-jawed holder, and on the other hand a holder which requires that the needle fit a cer-

tain notch in its jaws causes the wasting of much valuable time. Round curved needles flattened near the eye are very well controlled by a flat-jawed needle-holder. A beginner or one not wedded to some other variety will find the flat-jawed instrument represented in the accompanying cut (Fig. 286) very satisfactory. Some years ago the writer suggested that the notched jaws of a McBurney needle-holder be replaced by flat copper plates and that the Hagedorn catch be substituted for the simple spring; and the holder here illustrated is the result. The jaws are lined with heavy copper plates which render less liable the breaking of the needle and afford a firmer grasp. The spring catch is controlled by the little finger, and after a time one is unconscious of any effort to work it. It is made in three sizes and, with properly flattened needles, works very well. The most serious objection to this instrument is that the

Fig. 287.—Hæmostatic Forceps

Fig. 288.—Kelly's Fine-Pointed Hæmostatic Forceps.

copper plates wear out and must be renewed. If properly flattened needles were always at hand the copper plates could be dispensed with.

Hæmostatic Forceps.—Asepsis has made modern methods of hæmostasis possible. Formerly surgeons caught a bleeding vessel with a tenaculum or an anatomic forceps and, after carefully isolating it, tied a silk thread around it and left the end of the thread hanging out of the wound. In an aseptic wound it is not strictly necessary to isolate any but the larger vessels. Small bleeding points are grasped *en masse* and tied with an aseptic ligature, and the ligature is cut short. For this purpose we have hæmostatic forceps with scissor handles and a catch which, when locked, holds until loosened. This makes very rapid work possible without the loss of blood, as forceps can be applied and left until the operator is ready to tie the vessel. The temporary pressure made by the forceps will control the hemorrhage from most small vessels so that no ligature will be required. The exact shape of the forceps is immaterial, but it should not be too long or too heavy. It should have a small point and a reliable catch. They are usually made with a French lock and may

therefore be taken apart for cleaning, but, since they are always sterilized by boiling, the screw pivot is better because the French pivot soon becomes worn and loose and cannot be easily repaired. When the point is too large it grasps too much tissue and the artery cannot be so securely tied. An unnecessary mass of strangulated tissue is objectionable in a wound because it may become necrotic and the home of bacteria. Figs. 287 and 288 represent very good varieties of artery forceps. It is an advantage to have a few forceps curved at the end

Fig. 289.—Curved Hæmostatic Forceps.

Fig. 290.—Kocher's Forceps.

as represented by Fig. 289. A forceps that does not meet properly at the point, or which loosens after being applied to a vessel, should be repaired or thrown away, as it is unsafe. A few Kocher forceps of medium size (Fig. 290) are a great help in carefully holding the tissues, but are not as good as the other varieties for catching a bleeding vessel.

Ligature Carriers.—A ligature carrier is a very valuable instrument in abdominal surgery. Most of those in use are modifications of the Deschamps

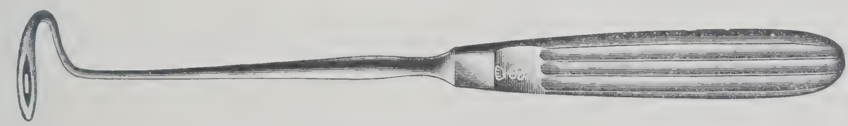


Fig. 291.—Blunt Ligature Carrier.

needle, the most important change being from the sharp point and cutting edge to a blunt point and edge. They are made right and left, but a surgeon usually needs but one (Fig. 291).

The Cleveland ligature carrier (Fig. 292) is a very convenient instrument. It economizes time and material because it has no eye to be threaded and because the ligature can be caught at the extreme end and is more likely to be tied without waste.

Retractors.—Retractors are often necessary, but they are also often used too much and too harshly. Many injuries have been caused by these instruments, when the patient was unconscious, that would not have occured during consciousness. A very important part of a surgeon' sduty toward his patient is to see that he is properly protected from injury of all kinds when helpless from

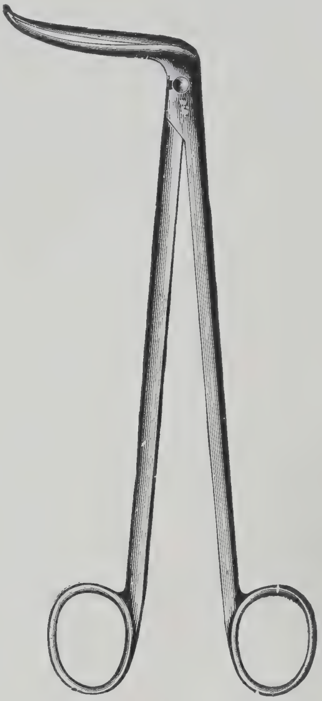


Fig. 292.—Cleveland's Ligature Carrier.

the anæsthetic, and one of the common injuries to which he is subject is that caused by improperly constructed or unskilfully handled retractors. The sharp-pointed retractors should rarely be used except when applied to some tissue which is to be removed, because they lacerate the tissues. Smooth retractors (Fig. 293) of various sizes will meet nearly every indication. The self-retain-

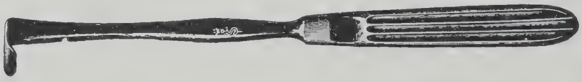


Fig. 293.—Langenbeck's Blunt Retractor.

ing abdominal retractor (Fig. 294) is a very helpful instrument. The amount of pressure made by it can be regulated by the one who applies it. It should be remembered that this or any other retractor will damage the tissue when applied with too great force or for too long a time. The special advantage of this instrument is that it takes the place of two hands, thus reducing the

number of assistants, which is always an advantage. The handles rest over the patient's thighs, out of the surgeon's way.

Nails and Screws.—Nails and screws have been extensively used in the treatment of fractures and after excisions, but they have not proven very satisfactory because, in spite of every precaution, they often prove troublesome. When allowed to protrude through the skin they are almost certain to cause local pressure necrosis and infection, which in turn lead to suppuration. When

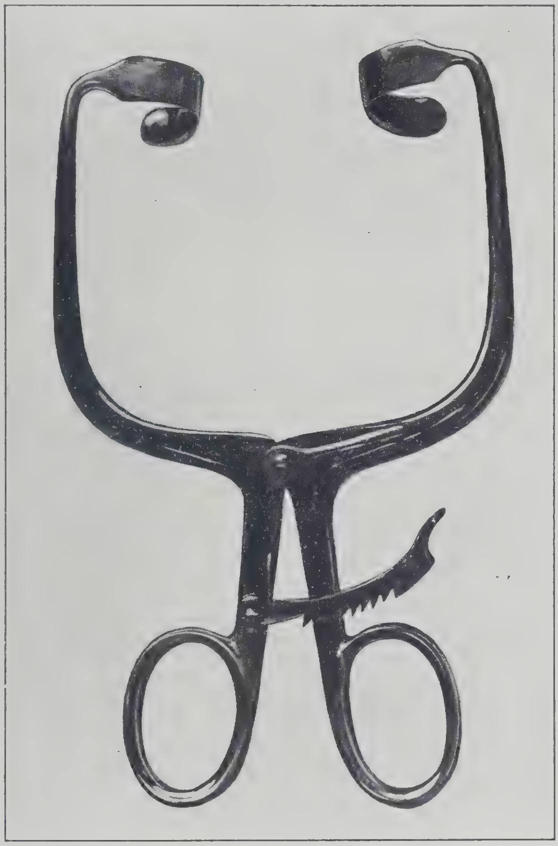


Fig. 294.—Self-Retaining Abdominal Retractor; takes the place of one assistant.

they are buried the results are more satisfactory, but even then they are prone to act as foreign bodies at a later period, causing necrosis and abscess, which necessitate their removal. As a means of approximating the bones after excision of the knee they have been superseded by chromicized catgut sutures. In the treatment of fractures they still have a limited field of usefulness, but even here the chromicized catgut suture is superior in the vast majority of cases because it holds the fragments more securely and becomes absorbed later. A nail or screw has in reality a very insecure hold on a bone because

it is only the outer compact layer which affords any special security, and even this hold loosens very soon. Fragments fastened in this way with seeming security will usually be found to have loosened after ten or twelve days. The elastic catgut allows of a little play from the first, but the elasticity does not increase during the life of the gut. The slender, small-headed steel nails and the screws of commerce are satisfactory for this purpose. They can be silver-plated at a trifling expense, and this renders them less liable to corrode or irritate. They should be sterilized by boiling.

APPLICATION OF THE PRINCIPLES.

Fads Not Principles.—During the development of our present technique fads have often been mistaken for principles. Certain things have been done that would have been better left undone. Poisonous powders and solutions were used in spite of the fact that their use was not justified by careful bacteriologic findings. As the truth has dawned upon us we have learned that good results were obtained in spite of these things rather than by virtue of them. Doubtless we have yet much to learn, but the fact that the technique has steadily grown more simple proves that we are freer from fads and nearer to principles. A scientific principle is based upon scientific facts and is capable of demonstration, but in surgery we have the element of life to deal with which often interferes with a demonstration of what clinically seems to be a fact. The real principles of surgery and of modern wound treatment, however, are scientific facts capable of demonstration. We are prone to run after fads because we have been much given to empiricism. Many of our fads have been excusable because we were doing many new things and results were constantly improving. The consequence was that some of the things done were given undeserved credit. It would seem a simple matter to eliminate a useless procedure by comparing the results obtained in a series of cases with this procedure, with those of another series in which the procedure was not used, but different observers often arrive at diametrically opposite conclusions from what are seemingly the same premises. Since we have scientific principles to work upon we should be slow to accept of anything based upon empiricism.

Bloodless Surgery.—This is a term which has a great attraction for the laity and for professional men who lack confidence in their ability to secure the aseptic healing of a wound. It received great impetus a few years ago when an eminent foreign surgeon came to this country to demonstrate his bloodless method for treating congenital dislocation of the hip. The so-called bloodless operation, however, with due deference to its real merit, is often bloody and attended with severe injury.

The subcutaneous tenotomy of superficial tendons is a good operation because the parts are superficial and the surgeon knows what he is doing, and

it leaves a small scar, but beyond this the field for bloodless surgery is limited in application.

Wounds.—The manner in which a wound is made has much to do with its healing. A wound made with a sharp instrument will heal more quickly and certainly than one made with a dull one. When possible an operation wound should always be made in the natural line of cleavage, passing either between muscles or through them parallel with their fibres. When muscles are split, the splitting should be done with an instrument sharp enough to separate the fibres without lacerating or bruising them. The conscientious surgeon will always handle the tissues of an unconscious patient as carefully as he would those of a conscious one. The preservation of the nerve supply is much more important than the preservation of the blood supply, because nature under all ordinary conditions very quickly restores the latter, while she often fails to restore the former, the consequence being atrophy and paralysis. Many ventral hernias are due to injury to the muscular nerve supply, not only from paralysis, but also from trophic changes.

The Treatment of Wounds.

In considering the application of the principles underlying wound treatment it is unnecessary to dwell upon the usual classification of wounds, but it is necessary to take up separately aseptic, suspected, and infected wounds, because there is a difference in the application of the principles under these different conditions.

Aseptic Wounds.—The treatment of an operative wound, which we take as a type of an aseptic wound, should begin hours or days before it is made, and end when it is completely healed—*i.e.*, when the stitches have been removed and the dressings are no longer needed. The stimulation of the patient's excretions, the cleansing of his skin, the preparation of the surgeon's hands, instruments, ligatures, and dressings, are all essential parts of wound treatment which enable the modern surgeon to secure primary healing. Old-time surgeons were better anatomists and often more skilled operators than modern surgeons, but the tyro in surgery, by the use of modern methods, can secure better results than the old-time surgeons dared to hope for, and can invade portions of the body then considered beyond the domain of surgery. This wound treatment has resulted in the transfer of many diseases formerly considered exclusively medical, to the domain of surgery. It is the outgrowth and travelling companion of the science of bacteriology. It is a promoter of modern pathology, because many important discoveries in pathology are based upon the knowledge of living tissues made possible by aseptic surgery. Pathologists groped through the abdomens of dead subjects for centuries without discovering that appendicitis, salpingitis, and gall stones are the causes of many pathologic conditions within the abdomen. Modern wound treatment has helped to raise surgery to a distinct science. If these facts be taken into consideration is it strange that

so much time is spent upon the teaching of the underlying principles of surgery, or that the practice of surgery based upon these principles attracts so many of the brightest minds of rising generations? One who enters the profession of medicine without a thorough knowledge of the principles underlying the treatment of wounds can never hope to compete with one who has that knowledge; and one who is careless in putting these principles into practice is sure to fall by the wayside.

Throughout the treatment of the wound the surgeon's efforts should be to assist nature in every way possible in her warfare against bacteria, and to give her such mechanical aids as will promote and supplement the natural biologic processes. The term primary union has no direct reference to the time required for union, but is used to designate an uninterrupted aseptic process from beginning to end. The time required for this process depends largely upon the perfection of the surgeon's work. Nature repairs the tissues by the interposition of a layer of new tissue of greater or less thickness, depending upon how closely the severed tissues are approximated; the closer and more accurately they are approximated the less the time required to complete the process. Every tissue is formed from cells of its own kind; and in order to secure perfect healing each layer of tissue must be closely approximated, muscle to muscle, tendon to tendon, skin to skin. Connective tissue, as its name implies, is found everywhere between tissues, and when unlike tissues are brought together they are united by connective tissue and the healing is not ideal.

The suturing of wounds is the most important mechanical aid to wound healing at the surgeon's command. Sutures have long been employed in surgery, but it is only since the introduction of the aseptic suture that the surgeon has been able to bring the opposite edges of each layer of tissue accurately together. In every wound of any considerable depth the tier suture by means of catgut, supported by stay sutures of silkworm gut or some other unabsorbable material, is an approved method of wound closure. Every careful surgeon takes ample time to close his wounds accurately because he knows that much of his success depends upon it. In closing an abdominal wound, for example, after perfect hæmostasis, the peritoneum should first be closed with fine catgut. Stay sutures of silkworm gut should next be introduced, beginning on the integument half an inch or more away from the wound and extending through all the tissues except the peritoneum. One stay suture for every inch and a half of wound is ample, and in short muscle-splitting wounds, as for an exploratory cœliotomy or an interval appendectomy, these sutures are entirely unnecessary. These stay sutures should not be tied until all other sutures are placed. The tendency at the present time is to abandon the through-and-through sutures altogether, but when they are properly adjusted the only objection to them is that they are made of unabsorbable material which must be removed. They are an extra safeguard against accident. They can be safely removed on the eighth or tenth day.

When muscles have been cut across they should be approximated by catgut sutures: but when muscle fibres have been separated by splitting they require no sutures, as the stay sutures are all that are necessary. The fascia of the external oblique is then carefully approximated by a running catgut suture, and next the integumentary wound is closed by a running lock stitch made of silk, horsehair, or catgut. Finally the silkworm sutures are tied. The tying of these sutures is important. The knot should never be over the wound, but at one side, and should not be too tight. All beginners and too many older surgeons are apt to tie stitches too tightly. Before they are tied both ends should be well pulled up so as to straighten out the thread, and then the knot should be tied tightly enough to give support to the buried sutures. These sutures should not be expected to do more than to act as a reserve support when for any reason union is delayed beyond the life of the catgut, or where an extra force is applied to the wound as in severe vomiting. When sutures sink deeply into the tissues so that the edge of the wound looks like the edge of a saw with blunted teeth, they are too tight and are very liable to cause pressure necrosis and abscess. On account of the slight swelling which follows every wound the tendency is for sutures to become tighter instead of looser, and there is no excuse for tying them too tightly.

Although catgut is not always a reliable suture material for an external wound, when but one row of stitches is used, it is coming more and more into use as an integumentary suture when the tier-suture method is employed. The borders of the integument when properly approximated adhere quickly, and when the wound has been closed without tension the finest catgut thread is excellent material for effecting a closure. The fine gut lasts long enough to perform its function, and does not have to be removed, which is a very great advantage. The iodine catgut seems to be well fitted for this purpose, as clinical experience and experiment prove that it is very difficult to infect. The running lock stitch or buttonhole stitch, made by looping the thread over the point of the needle each time it emerges from the tissues, is recommended by many because it is elastic, gives uniform support to the wound instead of making undue pressure in spots, and, when silk or horsehair is used, it is easily removed.

Stitch abscesses, at one time common in abdominal surgery, are rarely seen now. This improvement is not due entirely to a greater degree of cleanliness, but to other beneficent measures. They were caused by the through-and-through stitches which, unassisted, were given control of the tendency of the wound to gape, and which stitches were commonly tied too tight. With the continuous catgut suture applied to each layer, the through-and-through sutures have but little weight to carry, and there is no excuse for tying them too tightly. The bacteria in the deeper layers of the skin will cause no abscess except in the presence of necrotic tissue or of independent infection.

Before the wound is closed, chemicals in the form of solutions or powders should not be used, because they interfere with union. After closure the parts should be cleared of all blood by a piece of moist gauze and then thoroughly dried. Several layers of dry sterile gauze should first be applied, and over this should be placed a liberal layer of sterile absorbent cotton. The gauze quickly absorbs any moisture that may escape from the wound and keeps it dry. The cotton protects the wound from injury and excludes bacteria. In an abdominal wound, or in other parts of the body where a roller bandage cannot be accurately applied, the dressings should be held in place by adhesive strips, which not only keep the dressings from slipping and exposing the wound, but help to support the parts and prevent tension upon the stitches. For abdominal wounds the many-tailed bandage should be snugly applied and well pinned over the dressings. It should be carefully adjusted so that the pressure and support are everywhere uniform. It also helps to hold the dressings in place and to support the wound. When properly applied it is a great comfort to the patient. For wounds of the extremities and other parts of the body the roller bandage is preferable. A tight bandage is always a source of discomfort to the patient and is never necessary. In applying a dressing to a wound in the middle of an extremity it should never be applied so tightly as to necessitate the bandaging of the whole extremity to prevent swelling. In case of fracture it may be necessary to bandage the whole extremity because of the effects of hemorrhage and effusion underneath the skin. If the patient be a child it is often wise to apply a plaster-of-Paris bandage over the dressing. Dressings properly applied to an aseptic wound rarely need to be changed until about the eighth day, when the stitches are removed.

Rest is essential in the after-treatment of wounds, to avoid tension on the stitches. In large wounds this is best secured by rest in bed in a comfortable position. With the present methods of hæmostasis and wound-closing one has no fear of secondary hemorrhage and it is unnecessary to keep patients for a long time in one position. The careful changing of the patient in bed will not disturb the wound and will rest the patient, but a restless, nervous patient may do harm by causing tension of the borders of the wound. Rest of an extremity can be secured by its elevation, by frequent change of position, and by the application of a plaster-of-Paris or some other variety of splint. Since John Hunter published his famous dictum, that the first and great requisite for the restoration of injured parts is rest, physicians and surgeons have never ceased to be cognizant of its virtues. Rest as a therapeutic agent has been greatly abused in certain directions, and a reaction is taking place. This is specially true in surgery because we have learned that when a wound is once thoroughly healed the sooner the patient uses the part the sooner its function will be restored. Surgeons no longer keep their patients in bed as long as formerly after operations and injuries. The greatest abuse of rest has been in treating

sprains and tuberculous joints. The rest treatment of a sprained ankle continued for a series of weeks made it worse than a fracture. The prolonged rest of healthy knee joints for patients suffering from tuberculosis of the hip has without doubt caused in many cases bad results. It is the surgeon's duty to see that his patient gets rest when it is indicated, and to decide at the earliest moment when activity of body and joint shall begin.

Where there is loss of integument and for any reason skin grafts are not applied, a good way of dressing the raw surface is to use strips of sterile rubber tissue over the whole surface, like the shingles on a roof. This allows the escape of the discharges from the wound into the dressings and prevents the dressings from adhering to the wound. It is necessary to change this kind of dressing frequently because it soon becomes soiled and uncomfortable. A hand or foot, or a sterile raw surface, can be very satisfactorily dressed by applying sterile gauze directly and leaving it undisturbed for seven or eight days. The outer dressings can then be removed and the hand or foot with the adherent gauze immersed in a bowl of warm sterile water until the gauze loosens. This leaves a layer of clean healthy granulations. It is bad practice to remove adherent gauze from a wound by force, because it breaks down the granulations, hinders repair, offers a new entrance for bacteria, and causes the patient unnecessary suffering.

Suspected Wounds.—Every accident wound and every operation wound where aseptic precautions have not been observed, is open to suspicion and should be treated as if infection had taken place. The chances for successfully disinfecting a clean-cut wound are much better than in the case of a lacerated or contused wound, because in the latter there is more liability to infection. A good example of a clean-cut wound is an accidental cut across the palmar surface of the wrist caused by some sharp-cutting instrument. The hemorrhage, which will be severe, is the first consideration. It should be temporarily checked by pressure upon the arteries above and perhaps below the wound by fingers or bandage and compress. It is seldom justifiable to introduce an unsterilized instrument to stop hemorrhage. The surgeon should prepare himself and his instruments and get his dressings ready. He cleanses the hand and arm as if preparing for an operation, and finally cleanses the wound with sterile salt solution and a bit of gauze. The arteries are next tied with catgut, and the tourniquet removed. The median and ulnar nerves should be examined and, if severed, they should be united by fine chromicized catgut sutures. Two sutures should be passed through the distal and proximal ends of the nerve with a round-pointed needle, the ends approximated and the sutures tied. A few stitches should be placed in the connective tissue around the nerve in such a manner as to prevent undue tension upon the nerve sutures. The severed tendons should next be isolated and carefully sutured with chromicized catgut, great care being exercised that each end shall be sutured to its fellow. It will frequently be necessary to slit up the tendon sheaths to secure the proxi-

mal ends on account of muscular contraction. When this has been done the sheaths should be united by a running stitch of fine unchromicized catgut. When the wound has been made by a bright sharp instrument it can be safely closed after a careful cleaning. The superficial fascia should be closed by a running stitch of fine catgut, and finally the integument should be closed with silk or silkworm-gut sutures. A dry dressing of sterile gauze and cotton is then applied, and the hand held in a flexed position by a splint so as to keep the parts at rest in a relaxed state. If there is no unusual temperature this dressing should be left for one week, when the stitches are to be removed and a gauze dressing and the splint reapplied. As time goes by, the hand may be gradually extended so that it shall be straight by the end of the fourth week, which is about the time required for a tendon to unite. Should there be some elevation of temperature and local evidences of infection after two or three days, some of the superficial stitches must be removed and the wound treated as an infected one. Chemical disinfectants are not recommended in this class of wounds because an effort should always be made to secure primary union, and they would be more likely to prevent than to secure this result.

A lacerated and contused wound is usually an infected wound and should be treated as such. A compound comminuted fracture of the leg is a good example of this kind of wound. It is presumed that the arterial and nerve supplies are not injured to such an extent as to demand amputation. The hemorrhage, if free, should be promptly controlled by forceps and catgut ligature. The surgeon should first prepare himself, then the leg, then himself again, and finally the wound. The leg should be scrubbed, shaved, and prepared as for an operation. All infectious agents like pieces of clothing, etc., and all fragments of bone should be removed. When there is dirt in the wound the latter should be thoroughly cleansed with tincture of soap and warm water, aided by the fingers and a piece of soft gauze. Pieces of tissue that have evidently lost their circulation should be cut away. After it has been washed with soap the wound should be rinsed with warm sterile water, followed by a warm, weak solution of bichloride of mercury. It is useless to introduce the bichloride solution into a soapy wound. The fragments of bone should be adjusted and, when they cannot be held in position by splints and bandages, they should be drilled and fastened together by silver wire or strong chromicized catgut, preferably the latter. No iodoform or other powders should be put into the wound. Folded strips of rubber tissue or of gauze rolled in rubber tissue in the form of a "cigarette" drain, are so placed that they shall extend from the depths of the wound through the skin. They should not be large or numerous. Two will usually suffice. The wound should not be plugged with iodoform or other gauze, as it prevents drainage and healing. The pliable rubber tissue is better than rubber tubes because it drains as well and does not injure the tissues by pressure, as a stiff tube may. The wound when large should be partly

closed by loosely tied stitches of silkworm gut. It is not necessary to leave the wound wide open to secure drainage. The usual dry sterile dressing of gauze and absorbent cotton should be applied and the leg supported by a proper splint. The moist dressing should not be applied at first because it is often possible even in this class of cases to secure an aseptic wound, and the moisture would encourage bacterial development. It will be time enough to apply a moist dressing when suppuration is inevitable. This dry dressing may be left for four days unless there is a suggestive rise of temperature or the dressing becomes soiled. At that time, if it is apparent that an aseptic wound has been secured, the drainage material may be removed and a fresh dry dressing applied. If, on the other hand, the temperature rises or other evidences of infection appear, the dry dressings should be exchanged for moist ones and the wound treated as an infected one. So long as the wound remains aseptic it should not be irrigated or disturbed except for the removal of drainage materials and stitches, and the dressings should be changed only when the temperature rises or when they cause discomfort.

Secondary suturing of wounds is employed in aseptic wounds that have been drained and for the approximation of granulating surfaces. When drainage is employed where it is hoped that an aseptic wound may be secured, and the amount of drainage material used is enough to cause gaping of the wound, sutures should be introduced at the time of the first dressing and tied when the drainage material is removed. When aseptic granulating surfaces can be brought together without tension they will heal by first intention. Through-and-through sutures passing underneath the whole granulating surface should be employed when possible. It is useless to force the edges of a gaping granulating wound together with sutures under tension, because the sutures will cut through and do more harm than good. Under these circumstances it is better to approximate the surfaces with strips of adhesive plaster, supplemented by a comfortably fitting bandage.

Infected Wounds.—In preantiseptic days most wounds were septic and suppuration was so common that it was considered a necessary part of the healing process, and "laudable pus" was spoken of as something to be sought for. At the present time suppuration is known to be a pathologic process due to the presence of certain forms of bacteria in the wound, and when suppuration occurs in an operation wound the surgeon or some of his helpers may be responsible for it. In the preantiseptic days abdominal operations were very rarely performed because they were so commonly fatal, the patients dying from peritonitis. It was not unusual in those days for a medical student to go through his whole medical course, even where there were large surgical clinics, without seeing a single abdominal operation. At the present day peritonitis following operation is almost abolished and operations are often performed for the relief of that condition. When a wound has been infected it may finally heal by what

is known as secondary intention. In a suppurating wound the superficial layers of new cells formed by the tissues for the healing of the wound are destroyed by pus microbes and their toxins and they finally help to form pus. When this process is very active, the healing of the wound is interfered with. When, through Nature's efforts, and with the aid of the surgeon, a favorable change takes place, the pus becomes less virulent and diminishes in quantity. The new cells become more highly organized, and healing by secondary intention gradually takes place. The pathology of inflammation is partly based upon theory, and pathologists do not agree concerning suppuration, some claiming that it is purely pathologic, while others claim that it serves a useful purpose in combating invading organisms. The surgeon knows practically, however, that when there are no bacteria in a wound there will be no suppuration, no matter what the character of the wound or where situated, and his best efforts are therefore put forth to prevent infection and consequent suppuration. Aside from the dangers and loss of time which attend a suppurating wound, healing by second intention is unsatisfactory because of the large scar which it leaves. Accident wounds are suspicious wounds, and many of them do not come under the surgeon's care until after suppuration is well established. The principles connected with the treatment of these wounds are the same as those for the treatment of other wounds, but the details differ somewhat. When the evidences of infection—heat, pain, and redness of surrounding parts—are present, the surgeon's first efforts should be to allay the inflammation, the healing of the wound being then a secondary consideration. The discharges must be allowed free exit to prevent their absorption and dissemination from causing a general infection. If the wound is a deep one it must either be drained by tubes or strips of rubber tissue or be opened widely with the knife. It must not be packed with gauze, because gauze prevents drainage. It is a common error to pack a wound with medicated gauze under the mistaken idea that it will drain it. In treating infected wounds a rational use should be made of the best antiseptic and aseptic precautions; not that they will necessarily stop the suppuration, but that they may prevent the engrafting of another infection upon the one already existing. It is quite possible, for example, to inoculate a suppurating wound with the streptococcus of erysipelas or with the tetanus bacillus. Our knowledge of the exact relations of associated bacteria is quite limited. In suppurating wounds the infection is usually a mixed one, but there are very few instances known when the various forms of bacteria are at warfare with each other; on the contrary, the tendency is for them to unite their forces against the resisting powers of the tissues. Persistent or repeated irrigation of a suppurating wound with strong chemical solutions does more harm than good. It does not stop the suppuration and it is liable to injure the already weakened embryonic cells of the granulation tissue. Every experienced surgeon has observed patients with suppurating wounds which had refused to

heal under chemical irrigation, but which promptly improved after the irrigation had been discontinued. It is not practicable to use solutions strong enough or for a period sufficiently long to act as germicides, without danger of poisoning the patient. There is a growing belief that a claret-colored solution of iodine will accomplish much toward securing the desired result. Gentle irrigation with a warm normal salt solution meets every indication, since the only benefit to be derived from irrigation is a mechanical cleansing of the wound. The advantage of the salt solution over the sterile water is that it is more grateful to the tissues. A wound so situated and so shaped that it can be cleansed by gently touching it with pieces of soft gauze will heal more promptly without irrigation of any kind.

The poultice, so popular with the profession at one time and which still holds a prominent place in domestic surgery, has almost entirely given place to the moist gauze dressing. The poultice is an application soothing to an inflamed part on account of its warmth and moisture, but as usually made it is objectionable, and consequently it may become a source of danger.

Salves and ointments, at one time so extensively used, have fallen into disuse because they were found to be surgically unclean; however, they can be made in such a manner as to be a clean and comfortable dressing. Some of the heavier products of petroleum make the best ointments. When properly sterilized and impregnated with sufficient carbolic acid to prevent them from becoming infected, they make a very grateful dressing for a granulating surface. They can be spread upon gauze and applied directly to the wound. A granulating wound, which has become weak under rubber tissue or moist dressings, will often improve rapidly under the above dressing.

The moist gauze dressing has all the advantages of a poultice without its disadvantages. There is no clinical evidence that medicated gauze commonly has any advantage over simple sterile gauze. Surgeons who at one time used medicated gauzes quite exclusively now find that they can secure better results with the unmedicated gauze. The medicated gauzes are open to the same objections as powders and chemical solutions—weak ones may do no good, and strong ones may do harm.

After the wound has been gently cleansed it should be covered with a liberal layer of sterile gauze wet in sterile water. Very weak solutions of lysol and carbolic acid will give a perfume of their own to the dressings and are harmless, but they have no perceptible therapeutic value. An ample quantity of the moistened gauze should be applied, and over this should be placed a layer of rubber tissue to retain the moisture. The advantages of the moist over the dry gauze in this class of wounds are these: they do not adhere to the wound and they absorb the discharges more quickly. Over the rubber tissue a layer of cotton should be applied, and the whole held comfortably in place by a bandage. This dressing should be changed every three or six hours according to

the amount and character of the discharges. They should never be permitted to become dry. A chill or a rise of temperature indicates that the wound is not adequately drained, that there is systemic infection, or that there is some other focus or a complication, all of which are conditions that call for prompt attention. When the suppuration has been reduced to a minimum, the moist dressings should be replaced by dry ones and an effort made to secure rapid healing. When we have a granulating surface to deal with, it should be covered by strips of rubber tissue, and over the whole should be placed dry sterile gauze. When the wound is so situated or so shaped that the granulating surfaces can be approximated without tension, secondary sutures or adhesive plaster may be used for this purpose, provided the surfaces can be made sterile. It is only in exceptional cases, however, that this is possible, but it may be secured often enough to make the effort well worth while. For example, if an abdominal wound which presents clean granulating surfaces and has practically ceased suppurating be carefully dried, then thoroughly swabbed with ninety-five-per-cent carbolic acid followed by alcohol, then packed for forty-eight hours with gauze saturated with balsam of Peru, and finally closed by sutures or adhesive plaster, it will sometimes heal promptly, and when it does not heal at once the healing is at least hastened by this treatment. When, as sometimes happens, the rubber strips are uncomfortable, a very good and comfortable dressing can be made of gauze saturated in a mixture of six-per-cent balsam of Peru in sterilized castor oil.

A suppurating compound comminuted fracture of the leg may be taken as an example of this variety of wound. This is a particularly dangerous variety of wound because the soft parts are usually badly injured, and because there is danger of the burrowing of pus between the muscles. In addition, the medulla of the bone is exposed, and consequently there is imminent danger that a suppurating osteomyelitis may develop. The surgeon's hands, instruments, and dressings should first be prepared. The leg should then be shaved and scrubbed with soap, warm water, and brush. Finally, the wound should be thoroughly irrigated with a warm normal salt solution. If one who has been accustomed to use a strong bichloride solution in these cases will substitute a normal salt solution he will meet with an agreeable surprise. If the surgeon believes that a chemical solution must be employed, the iodine solution is probably the safest and most efficient. All parts of the wound must be reached and gentle pressure should be made along the leg from both distal and proximal ends toward the wound to ascertain whether there is burrowing. When the original skin wound is not large enough to admit of a free cleansing and ample drainage it should be enlarged, and when the wound does not permit free access counter-openings should be made. When burrowing is found, the pus pocket must be slit up or drained from the bottom. The wound should be gently but thoroughly explored with the finger, and if any loose spiculæ of bone or foreign

matters are found they should be removed. The fragments should be adjusted and held in place by extension made by an assistant pulling on the foot until the dressings and splint are applied. Rubber tubes or folded strips of rubber tissue, preferably the latter, should be passed to the depths of the wound and allowed to project from the wound for drainage. A large dressing of sterile gauze wrung out of warm sterile water should be applied. Over this a large sheet of rubber tissue and a layer of absorbent cotton should be applied and held in place by a snug-fitting roller bandage. A comfortable splint should then be applied with some mechanical arrangement by which free access can be gained to the wound for a change of dressings without disturbing the fragments. These dressings should be changed often enough to keep the wound clean and the dressings moist. A chill or rise of temperature always demands examination of the wound and perhaps a change of dressings. The latter should be continued until such time as the wound has so improved as to permit the application of strips of rubber tissue and a dry gauze dressing.

Drainage.—Drainage in some form has been employed since the days of Hippocrates. Old-time surgeons drained because they knew pus would form, later surgeons drained to prevent the formation of pus, and now we drain only when we have a suspected or infected wound or where there is unavoidable dead space. During the development of antiseptic and aseptic surgery drainage was a very common topic for discussion. Hippocrates first used drainage tubes for the treatment of empyema. Celsus and Galen used them for drainage in ascites. Ambroise Paré used gold and silver tubes. Heister first employed capillary drainage in the eighteenth century. At the beginning of abdominal surgery drainage was very extensively used, and for many years it occupied a prominent place. Surgeons then believed that peritonitis was only an exceptional cause of death, but that death was due to the absorption of what Keith called "that red serum, the enemy of the ovariotomist." Peaslee and Keith, in 1864, were the first to recommend peritoneal drainage through the vagina. About this time drainage through the rectum was tried; but from our present viewpoint we can readily understand why this proved fallacious. The soft-rubber tubes now in use were introduced by Chassaignac in 1859. Koeberle introduced perforated bulbous-ended glass tubes for peritoneal drainage in 1867, and soon after this Keith and Wells introduced straight glass tubes. These were all very popular for a time, but gradually fell into disuse because they soon became plugged and failed to drain. At that time drainage was considered a necessary part of the toilet of every wound, and more especially of an abdominal wound. Marion Sims was originally an earnest advocate of peritoneal drainage, but he was one of the first to recognize the fact that the opening of an abdomen does not necessarily indicate the need for drainage. With the development of antiseptic surgery surgeons learned that it is not the serum that endangers the patient, but the presence of bacteria. They also

learned that drainage is a source of danger because it serves as an entrance-way for bacteria, and therefore peritoneal drainage by tubes soon began to fall into disrepute. It was still believed at this time that peritoneal drainage was very often necessary, but surgeons began to realize that the tubes were not only dangerous, but that they were inefficient. Many varieties of tubes from many materials were tried, only to be discarded because of their dangers and inefficiency. Capillary drainage with gauze was next tried, but it was soon found that it will not drain pus and that it drains serum from the peritoneum for only a few hours, when its meshes become plugged and its capillarity is destroyed, and that furthermore it becomes adherent to the peritoneum. The gauze was then wrapped with rubber tissue to prevent adhesion, and this variety of drainage is much in vogue to-day. Prophylactic abdominal drainage was at one time given an extensive trial, but was found to be inefficient and dangerous. We have been guilty of many errors in the matter of drainage, and our sins of commission have doubtless greatly outnumbered those of omission, but we were obliged to go through this experience in order to learn what is necessary and what is unnecessary, and what makes for good and what for ill. All drainage openings become more or less infected, although all do not suppurate. The great trouble with peritoneal drainage, aside from its dangers, is that it does not drain. Experience and experiment have demonstrated that it is a physiological and mechanical impossibility to drain the peritoneal cavity for more than a few hours. Drainage from tubes ceases in about twelve hours and from gauze in twenty-four hours, because they are invariably walled off from the general peritoneal cavity in this time. There may be some flow of serum after this time, but it comes only from the drainage track and is caused by the presence of the drain.

The question of drainage is very important, and, unfortunately, it is impossible to lay down exact rules as to when we shall or shall not drain, because conditions vary with the patient, the environment, and the operator. From our present viewpoint much of the drainage done a few years ago was unnecessary and harmful, but it is quite probable that when surgeons employed it so extensively they needed it more than we do. It is very certain that in pre-antiseptic days it was very much needed. If at the present time one surgeon uses drainage where others do not find it necessary, it is quite possible that he needs it. The occasional operator undoubtedly needs it much more frequently than the regular surgeon, and it is certainly required in a very much larger percentage of accident than of operation wounds. Prophylactic drainage, or drainage to prevent the formation of pus, is no longer used.

The materials most frequently employed now are the soft-rubber tube of various sizes, rubber tissue, and gauze. The glass drainage tube, although in many respects excellent, is not so extensively used as it was at one time, because its length cannot be regulated as easily as that of the rubber tube.

The tube should be employed where large quantities of pus and large cavities or cysts are to be drained, as for empyema and for certain conditions of the urinary and the gall bladder. Folded rubber tissue is superior to the tube when it is applicable, because it is more flexible, is less likely to do harm from pressure, and does not cause gaping of the wound. This material is really very efficient, because it keeps the wound open enough without overdoing it, and the discharges escape along the side of the drain. For small accident wounds, and for larger ones where suppuration is not already established, the rubber strips are the best material. For capillary drainage, gauze is now the favorite material, although horsehair and silkworm gut are frequently used. Catgut and other absorbable materials have been disappointing as drainage materials on account of their tendency to become infected. Gauze is very commonly surrounded by rubber tissue to prevent it from adhering to the tissues; this is especially true in the peritoneal cavity. This combination of the two varieties of material affords both capillary and tubular drainage. The gauze drains the serum by capillary attraction, while pus and other heavy discharges escape along the side of the rubber tissue. This so-called "cigarette" drain should be made by laying a piece of sterile rubber tissue of the required size upon a table covered with a sterile towel, and upon this about four thicknesses of sterile gauze a little smaller than the sheet of rubber should be spread; then all are rolled rather loosely into a "cigarette." It should be made by the operating-room nurse when she is prepared as for an operation. This makes the best drain of the kind, as it is made of alternating layers of gauze and rubber tissue with the rubber outside, and affords drainage of both kinds. It does not adhere and can be removed at any time without causing pain or injuring the granulations. Capillary drainage with gauze is apt to be injudiciously employed. Medicated gauze should not be used as a drain because it has no advantage over plain gauze, and the drug may do harm. Sterile gauze, like every other drainage material, is a foreign body in the wound; therefore the minimum amount that will meet the requirements should be used, and it should be removed at the earliest possible moment. When gauze is used as a packing to control hemorrhage it may be necessary to use a considerable quantity and to leave it for from four to six days or until it loosens, but this is not drainage. The objections to gauze as a drainage material are these: it drains only serum and that only for a few hours; and when it is removed the act of removal may break down the granulations, causing pain and hemorrhage and furnishing a new entrance-way for bacteria. This is a particularly dangerous procedure in the peritoneum. Many lives have been imperilled by packing large quantities of gauze in the abdomen, and incautiously removing it on the third or fourth day. In superficial wounds the objections are not so potent, but the advantages are just as few. Surgeons have differed in regard to the use of gauze in the abdomen. When they have an abscess to open they carefully wall off the healthy viscera

with gauze before opening it because they know from experience that the pus will not go through it; then, after opening the abscess, they are apt to pack the abscess cavity with gauze, expecting the pus to drain out through it.

That drainage is often necessary all agree, but that it may be an evil none can deny. Many objections can be made to a drain, but the principal ones are: that it is an irritating foreign body; that it makes an entrance-way for bacteria; that it necessitates frequent dressings; that its removal may injure the granulations; and that it keeps the wound open, delaying the healing process. In the abdomen the presence of a foreign body interferes with the natural resistance of the peritoneum, and it sometimes predisposes to hernia, fistula, and intestinal obstruction. Indications for drainage can often be met in some other way. Strict asepsis renders drainage unnecessary in most cases. A peritoneum that has been carefully protected from injury during an operation will drain itself much better than one that is interfered with by the presence of foreign bodies in the shape of tubes and gauze. There are usually better ways of controlling hemorrhage than that of filling the wound with gauze.

Drainage is indicated in the presence of infection or where the chances are decidedly in favor of infection, and in the presence of much blood or cyst contents. As a rule, when the urinary bladder and the gall bladder are opened, drainage is indicated because they are usually infected before they are opened, and it is often necessary to make temporary provision for the escape of the contents. In large amputation wounds and after breast operations where the wound is closed, it is better to drain temporarily even when the wound is aseptic, because there is a large raw surface which in spite of the most careful hæmostasis will ooze, and serum will accumulate to fill the dead spaces—results which are unavoidable. When the lymphatics in the axilla, neck, or any similar regions are removed or destroyed by the operation, artificial drainage is very necessary for a time. These wounds will heal superficially without drainage, but the accumulation of serum and blood within will delay the healing of the deeper parts very much and may lead to secondary infection. An amputation stump should have two rubber tubes, of medium size, introduced one at each angle. They should not protrude from the wound so far that they may be bent over and obstructed by the dressings, which should be of dry sterile gauze and absorbent cotton loosely applied. A stout silk thread should be fastened to each tube and left protruding beyond the dressings, so that the tubes can be withdrawn without disturbing the latter; for every change gives the patient pain and may lead to secondary infection. They should be removed in forty-eight hours. Breast wounds should be drained in the same manner, save that it is usually better to make a stab wound through the integument at the most dependent part just large enough to admit a tube.

Drainage materials should be removed as soon as the discharges cease, for by this time they will have performed their function and will thenceforth only

act as foreign bodies. Where the surgeon hopes to be able to discontinue the drainage in a day or two, gauze should not be used unless it is surrounded by rubber tissue, for otherwise its removal may be dangerous and will surely be painful.

Abdominal drainage always causes peritoneal adhesions, but they may disappear after a time.

When a wound has been closed without provision being made for drainage, and a decided rise of temperature follows, the question of secondary drainage naturally arises. A temperature of 102° or 103° F. quite frequently occurs within twenty-four hours, but this is usually an aseptic temperature due to absorption of blood, and has no special significance. It often occurs with a simple fracture or with contusions where there is no question of infection. A temperature appearing on the second or third day and gradually rising commonly means wound infection, and in any event the wound should then be carefully examined. The peritoneum is the most tolerant and most capable of self-drainage of all the tissues; but when once this tolerance has been arrested and cannot be resumed, the patient dies. Secondary drainage of the peritoneum in such cases is practically useless.

Surgical Uses of Heat and Cold.

Heat and cold have been used as therapeutic agents in surgery from time immemorial. Their use has been largely empirical, based upon the fact that they relieve pain. Most of the statements concerning their effect are still empirical and difficult to prove. They are used rather indiscriminately for like conditions, heat being the favorite in winter and cold in summer, with seemingly like results. They have been favorites with the profession as well as with the laity in the treatment of inflammation in its various forms, but since we have learned that, surgically speaking, inflammation means the presence and activity of bacteria, our faith has been somewhat shaken, because any degree of heat or cold which will not destroy the tissues can have very little effect upon them. The mere presence of bacteria, however, is not all there is of inflammation. They must be active, and for their activity certain conditions of temperature and blood supply must obtain. It is these conditions which we hope to influence favorably by the application of heat and cold. Since we cannot hope to destroy the bacteria by these applications, our efforts must be to assist Nature in her warfare against them.

Clinical evidence concerning the effects of heat and cold is still conflicting and unreliable because observers have failed to give Nature due credit for what she will do without their aid. The more we learn concerning the conflict between bacteria and the living tissues the greater our respect for Nature's work and the more we realize that the most we can do is to be her faithful assistants. When inflammation is superficial it is rational to believe that we can render

some assistance by the judicious use of heat and cold, but when it is deep-seated it is difficult to understand how we can accomplish anything by their application. Many theories have been advanced concerning this matter, but none are proven. Writers will cite a long series of cases of appendicitis in which they have applied cold or heat to the abdomen with good results, and others will cite a like series in which they have kept their patients quiet in bed without applications of any kind and with equally good results. In the light of this evidence is it not rational to conclude that, aside from the relief from pain following the application of heat or cold, the improvement noted was due to Nature's efforts aided by rest?

In these few remarks on the surgical uses of heat and cold the writer does not wish to assume the rôle of an iconoclast, but he has not cared to voice theories which will not bear the test of intelligent clinical observation.

The influence which local applications can have upon the body temperature cannot be great and cannot extend to any depth, but they do add materially to the patient's comfort and are seemingly helpful when judiciously handled. It is probable that we can accomplish the most good through these agents by controlling the circulation of the inflamed part. The hyperemia which follows an infection is evidence that Nature is rallying her forces for the combat with the invading enemy, but when stasis occurs, the enemy has the advantage. If by the application of heat and cold we can facilitate the flow of blood through the part and prevent stasis we are certainly assisting Nature's efforts. These agents influence the circulation by their direct effect upon the superficial vessels and by stimulating the vasomotor nerves. That both heat and cold contract the small blood-vessels is demonstrated by their action in controlling capillary hemorrhage. The selection in each case must be left to the judgment of the attendant. When there is no definite indication for a choice it may safely be left to the caprice of the patient. In a few instances, however, a proper selection is very important. For example, when a hand or foot is so badly crushed that it is a question whether it can be saved or not, it would be a great mistake to apply ice, on account of its tendency to lower the vitality of the tissues. It would be a grave error to apply cold to an ulcerated cornea the vitality of which is low. Cold is the favorite in the early stages of inflammation when it is hoped that suppuration may be averted, but in the later stages, when suppuration is deemed inevitable, heat should take precedence.

Heat may be applied either moist or dry. When the skin is unbroken the choice may be made a matter of convenience, but, in the presence of a wound, no application should be made which is not in strict accord with the principles of aseptic and antiseptic surgery. Moist heat in the form of a poultice is not specially objectionable when the skin is whole, save that there are many more elegant methods, but when the skin is broken moist heat is highly objectionable, because in its usual form of a poultice it is crowded with bacteria and introduces foreign matter into the wound. Moist heat can be best applied by means

of sterile gauze wrung out of hot water and covered by rubber tissue. The dressings must be changed so frequently that they shall not become cold and dry, for alternating heat and cold are decidedly objectionable.

Dry heat is often just as efficient as moist and can be more conveniently applied by means of a rubber bag or coil. Dry heat has been extensively employed in the treatment of inflamed joints. The joint is surrounded by a metallic jacket, and the air within is gradually heated to the limit of endurance. This "baking" process is a very efficient means of relieving pain, but its curative effect has not been sufficiently demonstrated to gain for it an established place in treatment.

Extreme heat in the form of the actual cautery has an important, although limited, place in surgical treatment. It was formerly much employed as a means of controlling hemorrhage, but at the present time it is rarely used for that purpose. The use of the cautery is practically limited now to the treatment of hemorrhoids. The Paquelin cautery and the soldering irons are still used, but the electric cautery is the most convenient form of instrument. Modern operating-rooms are being provided with plugs so that the electric lighting current can be utilized for this purpose.

Cold is usually applied dry by means of an ice bag. Moist cold has no advantage over dry and is not nearly so convenient to apply.

PART V.

GENERAL SURGICAL PROGNOSIS.

GENERAL PROGNOSIS IN SURGICAL DISEASES AND CONDITIONS.

By LEONARD WOOLSEY BACON, JR., M.D., New Haven, Conn.

It is the business of *individual prognosis* to estimate the future course of any disease present in the individual patient, to answer the questions: Will this man survive this illness, this accident? If he survives, will his recovery be complete and permanent, will he go forth whole and sound, or will he always bear with him some disabilities resulting from his present condition of disease or injury, or some tendency to relapse? If he cannot recover, how long may he expect to live, or with what permanent disabilities will he be handicapped?

Passing beyond the consideration of the prognosis in the case of the individual patient, *special prognosis* considers the outlook of all those individuals as a group who are suffering from a particular ailment, and thus takes in, from the point of view of the several species of disease, the whole realm of medical and surgical nosology. That is to say, *individual prognosis* considers the outlook for A., B., and C., suffering, let us say, with appendicitis; while, passing from the individual to the group, *special prognosis* considers the wider subject of the outcome of appendicitis in its different forms and stages, and the results that may be expected under different forms of treatment, expectant or operative. In this way it is the function of *special prognosis* to consider all the units of the nosological schedule, and to determine and weigh the prognostic factors in cancer, erysipelas, aneurism, hernia, septicæmia, mechanical trauma, etc.

But, above and beyond all this, aside from the outlook for the particular patient, and aside from the several species of medical or surgical disease under consideration, are the general and fundamental matters of constitution and the powers of resistance.

Individual prognosis, the ultimate object of all prognostic study, depends, in the final analysis, upon the relation between the natural tendency of the specific disease with which the patient may be affected (the special prognosis) on the one hand, and the general powers of resistance of the patient on the other hand. These matters of constitution and the powers of resistance are the elements of "*general prognosis*," they appear as factors of prime importance in each and every case, and they are to be the theme of our study in considering

GENERAL PROGNOSIS IN SURGICAL DISEASES AND CONDITIONS.

It will be well for us, then, at the outset to indicate just what are these wide-reaching elementary conditions which affect the prognosis in all surgical diseases and in all surgical conditions, and then to proceed to examine them in more detail with regard to their bearing upon special and upon individual prognosis.

I. Age.—The most obvious of these general considerations is age. Infancy, childhood, adolescence, maturity, senility—all present factors whose prognostic import ranges throughout the whole field of medicine and surgery.

II. Sex.—A second matter is sex, though the influence of sex is more obvious upon the incidence of disease than upon its prognosis.

III. Constitution.—Many factors go to the making up of the constitution of the individual, and our study of this matter must be sufficiently broad to include that upon which our fathers laid great stress under the caption of temperament, and to include likewise a consideration of diathesis, heredity, and race.

IV. Integrity of Organs and Functions.—Integrity of organs and functions and the existence of concomitant disease will evidently demand a large share of our study, as including those factors influencing perhaps most closely the individual prognosis.

V. Environment.—Lastly, the environment of the patient will claim our attention, including under this term his occupation, his food, the climatic conditions under which he lives, his ability to create about himself hygienic conditions, and his disposition, through habit or training, to observe the laws of hygienic living.

Under these five heads—age, sex, constitution, integrity of organs and functions, and environment—we shall pursue the study of our theme.

I. Age.

(a) **Infancy.**—It is naturally in the extremes of life that we look for the influence of age upon surgical prognosis to be most pronounced. As indicated by Karewski,* a very energetic cell activity is characteristic of infancy and childhood. From this fact there result, however, two apparently contrary peculiarities, at once an enhanced and a diminished power of resisting noxious causes.

The intensity of metabolic processes in childhood occasions particularly favorable conditions for maintaining the conflict with micro-organisms and for the repair of trauma. We observe, indeed, that suppurative affections are relatively rare in children, and that solutions of continuity show a particularly favorable tendency to heal. In spite of the great frequency of abrasions and skin wounds, which are exposed to treatment quite opposed to the rules of mod-

* Karewski: "Die Chirurgischen Krankenheiten des Kindesalters," Stuttgart, 1894.

ern surgery, the surgeon sees far less frequently in the infant than in the adult progressive phlegmon or general sepsis arising from these sources. Even chronic suppurative processes, such as after puberty would be followed by rapid exhaustion, are astonishingly well borne and heal in a surprising manner. Certain chronic infectious diseases, such as tuberculosis and syphilis, seem to appear, as it were, in an attenuated form in children, and to proper therapy they yield much better results than in later life.

This same condition of enhanced metabolic activity results, on the other hand, to the disadvantage of the infantile organism. Inasmuch as the maintenance of this heightened activity is particularly dependent upon favorable and abundant local and general nutrition, it follows that all influences which acutely and considerably reduce this abundant nutrition are calculated to compromise the health or even the life of the child—a fact that applies to individual portions as well as to the whole body of the child. Impairment of blood supply and disturbances of innervation are the occasion of marked trophic disturbances, the former even occasioning the prompt supervention of necrosis, where in adults the same parts would have maintained their vitality. Indeed, a permanent depression of the general nutrition brings about a disposition to succumb to just those dangers against which the infantile organism is otherwise so particularly well fortified. Thus we understand how it is that even slight hemorrhage in nursing children may induce either sudden death or profound cachexia, that chilling of the body during operation or prolonged narcosis may be followed by fatal results, poisonous antiseptics bring about grave conditions, pyogenic infections assume a virulent and dangerous type. Conditions of malnutrition in children give a great impetus to the spread of micro-organisms, manifest, for instance, in multiple abscesses and miliary tuberculosis; while the very same cause occasions that characteristic trophic disease of childhood, rickets, which has its seat at the focus of the most energetic developmental activity.

These facts constitute so many indications for the regulation of our surgical treatment. While, on the one hand, they allow us within certain limits to count with more certainty than in adults upon good results in surgical treatment, and permit us to follow out, further than in adults, the trend of modern surgery toward conservative methods, on the other hand they warn us against protracted operations associated with loss of blood, and admonish us to watch narcosis with a jealous eye, to give special care to the selection of our antiseptics, and to modify in some instances our methods of operation.

These general principles will lead us to certain specific precautions in the surgical treatment of infants and children. When chloroform is to be administered, we must not allow too long an interval of fasting to precede anæsthetization, lest the patient begin the operation faint from hunger. Chilling of the body, or, indeed, of any portion of it, must be scrupulously guarded against, and for the same reason, viz., on account of the effect of high temperatures upon the

general nutritive processes of the child, no operations but those of urgency should be undertaken during extremely hot weather. Another wise prophylactic measure intimately affecting surgical prognosis in children, especially in hospital inmates, is, according to the suggestions of D'Arcy Power, to delay an operation of any magnitude, if it be possible, until the expiration of the incubation periods of those exanthematous diseases from which the child has not yet suffered. It must further be borne in mind that infants are extremely susceptible to interruptions and changes in diet, and that, except in the most urgent cases, it is well not only to correct any gastro-intestinal irregularities that may be present in an infant upon whom it is proposed to operate, but to make sure also, by actual trial, that the child can retain and digest the diet on which it is proposed to feed it after the operation.

Save in most urgent cases plastic operations are not best performed in early infancy. The diminutive size of the parts will, in many operations, add greatly to the difficulty of their execution, will enhance the difficulty of the exact hæmostasis which these operations demand *per se,* and which is furthermore exacted by the small body weight of the patient, and will make the procedure more prolonged and relatively more severe than in a somewhat older child.

Another matter to be thought of in connection with plastic operations, and indeed with any considerable operation in the infant, is the difficulty in applying and maintaining surgical dressings in the infant, and of keeping such dressings clean. In fact, any surgical operation demanding prolonged after-care must be considered relatively unfavorable in the infant; particularly does this apply to operations on or near the natural orifices of the body.

In cases where it is possible to provide properly for all these matters, there are, on the other hand, many advantages in surgical practice among infants. The ready depression of the vital forces in infancy is correlative with an equally ready recuperation. Furthermore, infants are relieved of the depressing effects of anticipation. As the infantile sensorium is relatively unimpressionable, they bear acute (*but not protracted*) pain relatively well, and they are not so likely to suffer from nervous shock. Many operations may be done on them without an anæsthetic, and the condition of semi-anæsthesia, so perilous in the administration of chloroform in adults, is relatively much less dangerous in infants. This is fortunate, because local anæsthesia, with cocaine, etc., is hardly applicable in infantile surgery.

(b) **Childhood.**—The general considerations which we have reviewed, as applicable to surgery in infancy, become less and less applicable as the age of the patient increases. The main distinction between infancy (say the first year of life) and childhood (say the ten years next succeeding) lies, from the surgeon's point of view, first, in a slight loss of the advantages of the energetic cell activity of infancy; and secondly, in a more than compensating gain in the stability of the nutritional processes of the older child as compared with those of the infant;

while, in the third place, there is a marked accentuation in the impressionability of the sensorium; and fourthly, the development of will power and voluntary action in the child come into active play.

This change from infancy to childhood affects surgical prognosis more particularly in the following ways: The intractability of spoiled children may so far interfere with examination before and after operation and hinder the carrying out of necessary treatment as to affect seriously the prognosis of the case. The impressionability of the sensorium being considerably heightened, nervous shock is more common and the patient is more likely to be favorably or unfavorably affected by his environment. While confinement to bed may be considered a quasi-normal condition for the infant, it is not so for the older child, and children bear confinement to bed and even to the house very badly. The relative immunity of the infant to certain types of infection grows less in the older child, and we find bone disease more obdurate; the tendency to diseases of the upper air passages (adenoid growths, etc.), with their deleterious effects upon nutrition, very marked; and a characteristic vulnerability of the lymphatics. Still, as compared with infants, children, when these obstacles can be met and overcome, are good surgical patients, and, in view of their comparatively ready response to medication, give us perhaps, all things considered, the best prognostic showing of any age.

The progressive increase of the child in stature brings with it, however, certain surgical restrictions which must not be lost sight of. The growth of the long bones takes place at the epiphyseal cartilages, and these must be respected. Typical joint resections are therefore inadmissible in infancy and childhood, and amputations in continuity will almost invariably be followed by a conical stump; while in bone disease involving the destruction of the epiphyseal cartilage, the prognosis as to the future development of the limb is distinctly bad.

While the pathogenesis of lardaceous or amyloid disease is not sufficiently well determined to enable us to assert a connection with the vulnerability of the lymphatic system which we have noted as characteristic of childhood, yet the fact is observed that chronic debilitating diseases, and particularly chronic suppurations, are prone to be followed by this sequel in childhood.

(c) **Adolescence.**—Three factors influence general prognosis in adolescence. They are, first, a supreme impressionability of the sensorium, so that at this age the *entourage* of the patient acquires increased prognostic import; secondly, the liability to blood dyscrasiæ, especially secondary anæmias; thirdly, a passing off of the relative immunity of infancy and childhood, so that diseases become more readily chronic, and chronic diseases, particularly bone diseases, exhibit a sometimes disheartening obstinacy.

(d) **Maturity.**—In considering the prognostic import of age as bearing upon patients in that period of life which we call maturity, full sexual development must be discounted in both sexes, and, with this, the liability of the patient to

chronic, perhaps concealed or unknown venereal infection. Sexual excesses and abnormalities have also an effect upon general prognosis. It is in this period of life that cares, responsibilities, and burdens rest most heavily upon us, and consequent neurasthenia, actual or potential, is of marked prognostic import. Chronic gastric or intestinal catarrhs may so affect general bodily nutrition as to compromise gravely a prognosis otherwise good, and the so-called "*dyspepsias*" are most frequent in middle age. The diatheses rise from latency to activity during this period of life, and alcohol, morphine, tobacco, or other drugs may be exercising a chronic depressing influence upon the vitality of the patient. The exalted activity of the cellular processes of infancy has spent itself, and the middle-aged man or woman has lost the attendant relative immunity of the infant and the child, but carries perchance instead the burden of many conflicts with invasive micro-organisms, which, while won, may yet have left in the patient depletion or debility as the price of victory.

(e) **Senility.**—Old age is the period when the various systems and processes of the economy begin to lose their normal balance, and atrophic and involutive processes give rise to certain characteristic conditions which are of prognostic significance. Of these perhaps the most important is the condition of the heart and of the blood-vessels, and the normal balance between their respective functions. We have seen how dependent upon a generous blood supply was the active cellular metabolism of infancy; scarcely less sensitive to the abundance and regularity of the blood supply are the tissues of the aged, in whom a constitutional condition bordering upon a dystrophy is to be expected, so that impairment of blood supply and disturbances of innervation react similarly upon the infantile and upon the senile organism. It is on account of their effect upon the nutritive processes of the senile tissues that chilling of the body during operations upon the aged must be scrupulously avoided; and so likewise any rough handling of the tissues by ill-conducted manipulation. Special pains must be taken in the aged to avoid increasing the intravascular pressure, for fear of apoplectic accidents. Although the aged bear hemorrhage badly, the same absolute loss of blood in ounces, inasmuch as it bears a less proportion to the total body weight, will probably be less injurious than in the infant. On the other hand, the diminished elasticity of the senile vascular system allows capillary hemorrhage to continue longer than in younger patients—a consideration of special moment in plastic operations and where large wounds are in question, with extensive flaps.

The bronchial and pulmonary conditions of the aged have an important bearing on the prognosis of such cases as may require a general anæsthetic, because the chronic bronchitis so prevalent in them, and the emphysema with which it is apt to be associated, are so readily exalted to the condition of broncho-pneumonia.

The diminished impressionability of the sensorium in the aged is a factor

favorable on the whole in prognosis. It diminishes the tendency to nervous shock, and it might even in some cases allow operations in the condition of semi-anæsthesia, recognized to be so perilous in any but the extremes of life.

Another characteristic senile change is of importance as regards surgical prognosis, viz., the atrophic condition of the skin. This affects seriously the chances of primary union in operative cases, and retards greatly the healing of accidental abrasions, contusions, lacerations, and other traumata involving the cutaneous and subcutaneous tissues. Its bearing upon the prognosis of extensive plastic operations in the aged is obvious.

Old people do not bear well confinement in bed. The development of bed-sores is hard to prevent, and they are of obstinate and often dangerous character when they occur. They are due to atrophy of the skin, to the absorption of the cushion of subcutaneous fat, to the degeneration of the blood-vessels, and to muscular debility preventing them from moving readily in the bed.

Yet senility is measured not alone by the years of the patient. Man has been said to be as old as his arteries, and the relative prognosis of a surgical case in old age will depend upon the degree of senile involution presented by the patient. Senile affections of the heart, the blood-vessels, and the lungs are the most important.

The aged offer but feeble resistance to microbial invasion, but under rigid asepsis modern surgery on aged subjects has furnished results surprisingly favorable, particularly in the surgery of those pitiable sufferers from prostatic hypertrophy, where ample drainage and careful asepsis have brightened the declining years of hundreds, whom a too timid distrust of the prognosis in surgical procedure on the aged would have abandoned to the miseries of "catheter life."

II. Sex.

As suggested above, the sex of the patient concerns more closely the incidence than it does the prognosis of surgical diseases. Yet many surgeons are convinced of the greater powers of passive endurance in women. This, indeed, is in conformity with a tendency which nature shows in the females of other animal species. A matter bearing weight in that direction, at least in this country, is the less addiction of women to alcoholism and other excesses too readily indulged in by men. To offset this is a proneness to chlorotic and anæmic conditions, which is more marked in women than in men.

But to pass from indefinite tendencies to more specific matters, we have to consider for a moment the effect upon prognosis of the arbitrary neutralization of sex by castration. Unfortunately the data available on this point are few, and I cannot do much more than to call the reader's attention to this as a possible factor in prognosis. Early castrates of either sex are rare. No statistics, so far as I know, are available as to the general powers of resistance of oöphorectomized women as compared with those of their more fortunate sisters.

Though we are not considering the prognosis of the operation of castration *per se,* which belongs to the domain of special as opposed to that of general prognosis, yet it is to be observed that the importance of the generative glands upon the general vital processes has been made strikingly evident since the studies provoked by Brown-Séquard's experiments (with orchitic extracts), and more recently by the ill effects attending castration as a relief for "prostatism" at an age when the generative glands might be supposed to have but a diminished influence upon the general metabolism of the individual.

What I have to offer further upon this subject of the relation of sex to surgical prognosis concerns exclusively the sexual functions of women—menstruation, pregnancy, abortion, parturition, lactation, and the phenomena of the climacteric; and the consideration of these questions will lead us to adopt a reciprocal method of discussion, one to which we shall have frequent occasion to return in the course of this study, viz., the investigation, first, of the impression of these several functions upon the course of surgical diseases; and then, reciprocally, the impression exercised by surgical procedures upon these several functions.

(a) **Menstruation.**—As to menstruation, if the old theory of its being the result of a general plethora had been true, it might have been conceived to have a favorable prognostic value with regard to the nutrition of the tissues in connection with surgical operations; as a matter of fact, however, menstruation, when normally performed, has but slight influence upon prognosis. This much, however, may be admitted, that with normal menstruation it is not uncommon, in cases of critical illness, to see improvement apparently stimulated on the appearance of the menstrual flow.

It has been my habit, in operations of election upon women, to choose a time a few days after the close of the menstrual period, with a view of eliminating any possibly unfavorable effect of menstruation as a complication, till such a time as the patient might be expected to be beyond the critical stage of recovery; and this in operations other than those upon the reproductive organs. In gynæcological operations proper this plan would seem to be the more entitled to consideration.

On the other hand, with regard to the reciprocal effect of an operation *per se* upon the function of menstruation, this may be said: that the effect of a major extra-genital operation is variable and can hardly be predicted. It may hasten menstruation by a few days (perhaps the most common effect), or indeed precipitate it immediately; it may retard its appearance by a few days or weeks, or indeed cause its suppression for one or more periods; or (as frequently happens) it may not exert any traceable effect upon that function.

(b) **Pregnancy.**—The question of pregnancy in its effect upon general surgical prognosis is as hard to estimate as that which we have just been considering. In early pregnancy the intractable vomiting which this condition some-

times induces may be a factor of great importance. In advanced pregnancy the tendency to eclampsia merits consideration, particularly in primiparæ, and so likewise the interference with proper respiration occasioned by the mass of the pregnant uterus in the abdomen.

In operations upon the abdominal wall the severe strain upon the cicatrix must be borne in mind, especially in operations for ventral and umbilical hernia.

Strangulation of inguinal and femoral herniæ, due to the pressure of the gravid uterus, does not seem to occur with any such frequency as might *a priori* have been expected, especially when we consider the difficulty or even impossibility of retaining the herniæ with a truss during the latter part of gestation. As to the effect of pregnancy upon neoplasms connected with the reproductive organs, whether the breasts or the pelvic organs, it is well known to cause a great acceleration in their growth. Pregnancy occasionally will greatly retard the process of calcification in the healing of bony fractures, and it is said that newly healed fractures may even lose their recently deposited calcium salts upon the supervention of pregnancy. The disastrous effect of pregnancy upon osteomalacia has been repeatedly observed. The debility arising from too frequently recurring pregnancy can best be considered as a phase of neurasthenia.

The reciprocal of the question of the effect of pregnancy upon surgical diseases is that of the effect of surgical diseases and operations upon pregnancy; that is, the likelihood of their producing abortion. Upon this question some interesting and instructive data are available, showing us the remarkable tolerance exhibited by the pregnant uterus in the face of operative procedures of considerable magnitude, even when directed to the reproductive organs, to the immediate uterine appendages, and, *mirabile dictu*, to the uterus itself.

Thus Gordon * relates the case of the removal of a sessile uterine fibroid, nearly as large as the uterus itself, done at the third month of pregnancy, without interrupting the pregnancy or apparently affecting delivery at the completion of the period of gestation; and Sylvester † removed, in the third month of pregnancy, a uterine fibroid, weighing eight and three-quarter pounds and having a pedicle two inches in diameter, with normal delivery of a living child at term.

Breast amputations are related as late as the sixth and seventh month, also trachelorrhaphy; and one man ‡ vouches for the dilatation of the *internal os* (sic) for the relief of hyperemesis, all without causing abortion or miscarriage. Oöphorectomy has been repeatedly performed during pregnancy, and, according to A. P. Dudley,§ if it causes miscarriage it does so only by hemorrhage *into* the uterus, which on this account should be scrupulously guarded against. Yet the

* Gordon: Trans. Maine Med. Assoc., 1889, vol. 10, pp. 99–104.

† Sylvester: New England Med. Gazette (Homœop.), Boston, 1890, vol. 25, p. 397.

‡ Trans. Maine Med. Assoc., *loc. cit.*

§ Trans. Maine Med. Assoc., *loc. cit.*

fact remains that a considerable number of abortions do occur after even extragenital major operations, and that pelvic and abdominal operations are particularly likely to induce this accident. This may be due in part to the death of the fœtus by intoxication from the anæsthetic rather than to the operation *per se;* hence the recommendation to elect ether in preference to chloroform in these cases, as being less noxious to the fœtus.

(c) **Parturition.**—The prognostic importance of parturition relates to its effect upon certain few surgical conditions, of which hernia on the one hand, and aneurisms and varices and certain ocular and cerebral conditions on the other hand, are perhaps the chief. These have in common an intimate dependence upon the effects of the powerful contraction of the uterine and abdominal muscles, and the resultant intra-abdominal pressure, either directly, as in hernia, or, as in the other conditions, through the effect of these voluntary and involuntary muscular contractions upon the blood pressure.

In labor, as during pregnancy, though the effect of these factors upon hernia must be regarded as unfavorable, yet accidents of strangulation are very rarely reported in medical literature. For aneurisms and varices, as well as the other ocular and intracranial conditions alluded to, the peril may be said to be directly proportional to the blood pressure, and may make the early use of anæsthesia and the prompt resort to instrumental delivery imperative. It is the province of special prognosis to consider the outcome of the parturient act itself, and likewise to take cognizance of its effect upon the uterus and the perineum, which may or may not have been the seat of previous plastic operations.

(d) **Lactation.**—Lactation, except the effects of too prolonged lactation which are substantially those of neurasthenia, is of prognostic importance chiefly in proposed operations upon the breasts. Their marked increase of blood supply during this period makes the danger of troublesome hemorrhage somewhat greater.

(e) **The Climacteric.**—As to the effects of the climacteric upon the general nervous system, they are likewise substantially those of neurasthenia, and but one important factor concerns us here, viz., the effect of the menopause upon neoplasms. The only case in which its advent can be considered to have a favorable effect is in that of uterine fibroids. In this case, while undoubtedly a considerable number of tumors of that class cease to be troublesome after the establishment of the menopause, yet this advantage is greatly offset by the tendency of these neoplasms, as, indeed, of many other forms of new growth, to undergo malignant degeneration at this period of life.

III. Constitution.

In a certain sense the term "constitution" is the summation of all the elements of our present study, as age, sex, disease, etc., are but factors making up the constitution of the individual. In a more limited sense, however, we may

consider the constitution of the individual to be that original fund of vitality and capacity to resist and overcome disease with which his temperament, his diathesis, his personal heredity, and his race have endowed him.

(a) **Temperament.**—The consideration of temperament has so far gone out of fashion that even the terms in which it was discussed are now no longer comprehended. The conception of the four principal temperaments—the sanguine, the nervous, the bilious, and the lymphatic—was doubtless the product of more or less fanciful reasoning upon erroneous data of the old humoral pathology. Nevertheless, there are certain mental, moral, and physical characteristics which present themselves in certain groups of individuals, and these, taken as a whole, have an appreciable influence upon such surgical diseases and conditions as may affect the members of that group. The sanguine temperament, fair-haired, blue-eyed, with energetic movements, may not in its pure type have a special prognostic coefficient of its own, yet it is possible to select, according to some more or less vague criterion, groups of individuals of whom, though they be all at the moment in equally good health, it will be possible to predicate that these, by temperament and constitution, have a better chance to recover than have those from equally grave surgical diseases and conditions. In these, call their temperament sanguine or nervous, as you will, we can count upon the patients to co-operate with the surgeon to achieve their own recovery—they make "good patients"; while in those it is recognized that, call their temperament what you will, bilious or lymphatic, the prognosis is relatively unfavorable, and they may succumb from sheer inability to summon the forces of recuperation. The first have warm extremities, active circulation, and energetic movements; the second have cold hands and feet, muddy or pale complexion, and sluggish movements. However vague these conceptions are, they have a certain undeniable prognostic weight.

(b) **Diathesis.**—The vagueness of our conception of temperament is slightly relieved when to it we add the likewise indefinite conception of diathesis. A proper mingling of temperament and diathesis gives us the well-recognized type known as the "habitus phthisicus." Now, though we are dealing with confessedly shadowy and indefinite quantities when we discuss temperament and diathesis, yet a little attention will bring to the fore one important rule of prognosis, to wit, *where the incidence of disease corresponds to well-marked temperament and diathesis, the prognosis is relatively unfavorable.* For instance, the concurrence of surgical tuberculosis and the "habitus phthisicus" adds greatly to the gravity of the prognosis; the "dartrous" diathesis, coupled with the sanguine or the lymphatic temperament, together constitute a type in whom ulcerative processes will be found exceptionally obstinate; those of bilious temperament and of the gouty diathesis will have calculous troubles of greater gravity than will members of the other groups, should the inciting factors of calculus formation present themselves. Yet ulcers heal kindly in those of the

phthisical habit; those of the sanguine or the lymphatic temperament and with a tendency to dartrous manifestations are less likely to have grave symptoms from calculi; and those of bilious or nervous temperament with a gouty diathesis are so far immune to tuberculosis that if it should establish itself the prognosis is far better than in those of the first group.

Our modern knowledge of the rôle of micro-organisms in the causation of disease leads us to overlook much that was of value in the keen observations of our fathers. So much have microscopy and the so-called "laboratory methods" of diagnosis occupied the modern physicians, that little scientific study has been devoted to the analysis, delimitation, and classification of diatheses. There is vaguely recognized by the profession of to-day a gouty (or arthritic or lithæmic) diathesis, and the studies of Bouchard tend to group this and certain other morbid tendencies under the head of troubles due to defective oxygenation or hypometabolism. It seems to be acknowledged that the gouty diathesis is likely to entail upon its subjects arteriosclerosis and chronic nephritis, and these would have a decided prognostic significance in surgery, inasmuch as temporary urinary inadequacy may easily be a sequel to anæsthesia or even to simple surgical shock. One other diathesis has acquired tolerably distinct recognition, viz., the neuropathic; and it is likewise possible to establish as a fairly distinct entity the obese diathesis. One important bearing of this latter upon surgical prognosis is with reference to its effect upon the heart, whose function may be greatly impeded by massy deposits of fat and by fatty infiltrations of the myocardium. A second consideration is the thickness of the subcutaneous panniculus, with its heavy mass of tissue of low vitality, prone to suffer the invasion of pyogenic germs. A third is the possibility of fat embolism, when it is necessary to work in great adipose masses, *e.g.*, in deligation of the omentum.

In general, good bodily development, large bones, hard muscles, little fat, yet good body weight, and a history of freedom from previous disease—these are the indications of a good constitution, irrespective of temperament or diathesis.

(c) **Heredity.**—When we pass from the consideration of temperament and diathesis to that of heredity, we come upon a more stable footing, and, though the subject is one of infinite complication and dispute, yet certain facts are sufficiently clear to have a distinct bearing upon prognosis. For the most general of these facts concerning heredity we are indebted to the observations and records of life-insurance examiners, who have demonstrated the highly influential distinction between long-lived families and short-lived families. The effect of this hereditary characteristic upon surgical prognosis is very obvious, and at the same time very considerable.

Of secondary importance in their influence upon surgical prognosis are syphilis, rheumatism, gout, tuberculosis, alcoholism, obesity, diabetes, nephritis, cancer, insanity, and epilepsy in the parents. Although the common effect of all these parental diseases is to impart to the offspring a weakened constitution,

yet bad heredity for one disease may not be bad heredity for all; *e.g.*, a gouty heredity is rather favorable than otherwise in the prognosis of surgical tuberculosis.

Heredity may determine, however, more than a mere feeble resistance to disease. It may determine a distinct *locus minoris resistentiæ*, stomach, liver, uterus, and skin showing respectively an inherited tendency to the localization of disease upon these particular organs.

The heredity of malignant diseases may come under both these categories; *i.e.*, the transmission of the tendency to specific forms of disease, and the transmission of the tendency to the involvement of a particular organ. One question which suggests itself in this connection I am not able to answer for want of statistics, viz., whether malignant disease shows an enhanced malignity in those predisposed by heredity as compared with those not so predisposed.

We have stated it above as a law that the concurrence of temperament and diathesis with the incidence of disease is markedly unfavorable upon prognosis. When to this triad there is added a fourth factor, viz., heredity, the prognosis becomes still more unfavorable; as is illustrated in the case of tuberculosis, where, when this disease fastens upon one of the phthisical habit, a tuberculous family history adds appreciably to the gravity of the prognosis.

The most patent illustration of the effect of heredity upon disease is seen in the case of hereditary hæmophilia, with its well-known transmission of hemorrhagic tendency through the female line.

(d) **Race.**—The summation of hereditary influences is to be seen in the problems of race. Surgical prognosis in mulattoes, as indeed in most mixed races, is certainly relatively unfavorable.

Concerning the African negroes of full blood, it may be affirmed that, at least in the savage state, their less highly developed nervous system renders them comparatively insensible to pain and shock. Bordier * is the authority for the statement that among the Yoloffs it is a not uncommon practice to rip the abdomen open and handle the protruded bowels, with a view of testing the virtue of the "*gri-gris*" given by an itinerant marabout, and then return the exposed entrails into the abdominal cavity with apparent unconcern. This is surely convincing not only of the lesser sensibility of these people to pain, but also indicates a greater immunity from the usual dangers of peritoneal infection. Similar facts can be quoted with regard to aboriginal tribes of this country, for instance, the Modocs. This combination of circumstances—*i.e.*, a naturally diminished peripheral sensibility, coupled with a more passive condition of the mind—makes the negro a most favorable subject for all kinds of surgical treatment with or without preliminary anæsthesia. Similar testimony is offered by medical missionaries concerning the Asiatics of the Pacific littoral.

* Bordier, quoted in article by Matas on "Surgical Peculiarities of the Negro," in Dennis's "System of Surgery," vol. 4, p. 847.

The greater immunity formerly enjoyed by the negro in respect of certain diseases is rapidly disappearing, and he now not only shares the physical weaknesses of the white race, as exhibited on this continent, but is rapidly developing previouslyunknown predispositions, which are increasing his general tribute to disease and death even to a more fatal degree than in the white race.* In one particular we must consider further the influence of race upon surgical prognosis, viz., with regard to plastic surgery in the negro. The racial tendency to keloid growths in cicatrices must always be borne in mind in planning operations of this description upon negro subjects.

IV. Integrity of Organs and Functions.

In considering the general prognosis of surgical diseases and conditions, great stress must be laid upon the integrity of organs and functions, and on the existence of concomitant disease. This important factor meets us in all questions of surgical prognosis, whether we are considering treatment, or exploration, or the mere duration of life in a hopelessly compromised case. Evidently to consider this division of our subject with anything bordering on exhaustiveness would call for an elaborate review far exceeding the limits of our chapter, and we can touch here only on certain limited phases of the subject. Instead of taking up seriatim the maladies from which the surgical patient can suffer in any of his organs, I propose to consider but a few of these, and to consider them in their application, first, to anæsthesia, and secondly, to surgical operations proper, leaving the rest to be considered elsewhere in this work, under the special prognosis of the several surgical diseases, conditions, and procedures there discussed, or considering them to have received already sufficient general consideration in the preceding paragraphs.

The first part of the subject may perhaps be most simply considered in an attempt to answer the question, What morbid conditions render the administration of a general anæsthetic especially dangerous? while, for the second part, we shall attempt to answer the question, What are the morbid conditions which especially enhance the dangers of surgical operations *per se?* Some few questions properly concerning general prognosis which do not come under this scheme we shall touch upon briefly at the close of this section.

1. What Morbid Conditions Render the Administration of a General Anæsthetic Especially Dangerous?—In many surgical procedures—indeed, it is safe to say in the majority of aseptic operations, major and minor—the chief danger to the patient lies in the administration of chloroform or ether for the purpose of anæsthetization. It is well known that certain diseases of the heart and blood-vessels, certain diseases of the kidneys, certain diseases of the lungs and bronchi, and certain morbid conditions of the nervous system render anæsthetization extra-hazardous. Let us examine these facts more closely.

* Matas, *loc. cit.*

(*a*) *Cardiac and Vascular Diseases.*—The chief dangers in anæsthesia come from sinking of the blood pressure. The causes which occasion this are obviously three—acute hemorrhage, cardiac insufficiency, and general or extensive local vasomotor paresis.

So obvious is the effect of acute hemorrhage that we need do no more than mention it. It is within the experience of every anæsthetist.

With regard to cardiac insufficiency, it may be said that, contrary to earlier teaching, fully compensated valvular heart disease is not so much to be dreaded by the anæsthetist as to call for anything more than a little extra care in the administration of the anæsthetic. Valvular disease with under-compensation is a condition of greater gravity, though, except in cases of aortic stenosis, the situation is measurably relieved by the depletion afforded by the operative incision.

The actual functionating of the organ is, after all, what counts, and a heart acutely dilated by overexertion, or constantly overstimulated by alcohol, or driven to undue frequency of rhythm by thyroidal or sympathetic disturbances, or weakened in its myocardium by the toxins of acute disease, presents perils for anæsthesia quite as considerable as those attending an obstructive or regurgitant lesion of the cardiac valves. It does not appear that mere cardiac arrhythmia, such as follows chronic abuse of coffee or tobacco, the "irritable heart" which causes so much distress and alarm to its subjects, is of great concern in the prognosis of anæsthesia, though it may well be that the well-recognized perils of semi-anæsthesia, through its tendency to *inco-ordination of the reflexes*, are greater in those subject to these functional troubles of the heart.

The most exquisite picture of general vasomotor paresis is seen in surgical shock, a condition in which general anæsthesia is universally recognized to be extremely perilous. The syncope of nervous women, the tendency to faint at the sight of blood or pain, and even a history of habitual syncope from slight causes are not, when unaccompanied by other evidences of cardiac or vascular disease, of great prognostic importance as far as concerns anæsthesia; and this because they are rather the expression of an unduly impressionable sensorium than of a morbid vasomotor apparatus, and it is just the office of anæsthesia to abolish the sensorial impressionability, and thereby the dangers from this source are *ipso facto* eliminated.

A much more important consideration is calcareous degeneration of the arteries, whereby they lose their ability to adapt their calibre to the varying demands of their blood content.

(*b*) *Renal Diseases.*—Standing in an intimate and sometimes causal relation to the affections of the heart and blood-vessels are diseases of the kidneys, and, in connection with the prognosis of anæsthesia, these must be considered in their reciprocal relation; *i.e.*, both the effect of kidney disease upon the cardio-vascular system during the comparatively brief period of surgical anæsthesia, and, *per contra*, the effects of anæsthetic drugs upon the diseased kidneys. It is

for lack of examination of this subject from these two points of view that we find such divergent and conflicting opinions with regard to the relative safety of ether, chloroform, and other anæsthetic drugs.

In considering the effect of chronic kidney disease upon the immediate prognosis of anæsthesia, *i.e.*, upon the liability of the patient to sudden death during or shortly after the operation, we observe that the *modus nocendi* of chronic kidney disease is not through defective elimination of the ordinary urinary toxins, for even total suppression of renal function may continue some hours or days without fatally compromising the life of the individual; nor yet in the defective elimination by way of the kidneys of the anæsthetic which is being administered, as we have abundant experimental proof that a large quota of its elimination is through other channels (lungs, stomach); but rather through the direct effects of chronic nephritis upon the cardiac muscle and upon the walls and the vasomotor apparatus of the arteries. The well-known chronic exaltation of blood pressure fatigues the hypertrophied and diseased heart, there is no reserve fund of cardiac strength to draw upon, and the adaptive function of the vasomotor apparatus is chronically inoperative; hence sudden death under either chloroform or ether.

Viewed from the opposite standpoint, we must consider the anæsthetic in the line of an irritant drug, similar to turpentine or cantharides in its effect upon the diseased kidney. When proper weight is given to this aspect of the case, it is my opinion that the dogmatic claims regarding the superior safety of ether over chlorofrom must be revised. Ether may yield fewer cases of sudden death during or shortly after anæsthesia, but in the subjects of cardiac, vascular, and renal diseases it is probably answerable for a considerable proportion of those melancholy cases where we are told that "the operation was successful, but the patient died."

(*c*) *Diseases of the Respiratory Organs.*—Concerning the relation of diseases of the respiratory organs to the prognosis of surgical anæsthesia, we may be more brief, and we may summarize the situation by saying that in conditions of subacute and chronic inflammation of these organs, the irritant effect of anæsthetic drugs administered by inhalation must be accorded a considerable prognostic weight.* Insufflation pneumonias are easily induced and add largely to the peril of anæsthetization, especially when there is vomiting on the operating table. Particularly dangerous in this regard is the putrid and fecal vomit of obstruction to the alimentary canal, whether acute or chronic.

2. What are the Morbid Conditions which Especially Enhance the Dangers of Surgical Operations "per se"?—It will be seen that when we have disposed of those affections in which the dangers are chiefly from the anæsthetic, we have

* In the presence of latent bronchial and pulmonary inflammations, the possible advantages of the administration of ether or chloroform by the rectum might merit consideration, and so likewise the merits of "spinal anæsthesia" from subdural injections.

greatly limited the scope of our present inquiry; nevertheless, it will behoove us to pass briefly in review the diseases of the various organs, with particular reference to their effect upon the prognosis of surgical operations *per se*.

(*a*) *Cardiac and Vascular Diseases.*—With regard to the affections of the circulatory system, it may be said that valvular heart disease, especially if undercompensated, has a great effect upon the nutrition of the tissues, and interferes greatly with the capacity of the organism to protect itself against microbial invasion. On the other hand, once the operation is overpast, the rest in bed, which is demanded by the after-care of many surgical procedures, may act most favorably upon a cardiac lesion. Endarteritis is of prognostic importance, in that it compromises seriously the nutrition of the tissues. Its influence is most markedly unfavorable in the treatment of gangrene and aneurism. The presence of phlebitis is of great moment, even if it does not affect directly the part to be operated upon, because it involves a possibility of extensive thrombosis, embolism, and, in the presence of sepsis, of pyæmia.

Marked hereditary hæmophilia raises even the most trifling operations to a rank of extreme peril.

(*b*) *Renal Diseases.*—Few surgeons will face with any satisfaction the necessity for operating upon parts œdematous from chronic renal disease; and this aside from the dangers connected with anæsthesia, on account of the extreme liability of these œdematous tissues to pyogenic invasion. One weighty counter-indication to kidney operations must be borne in mind, viz., the possibility of the existence of but a single kidney (once in twenty-four hundred cases, Morris).

(*c*) *Diseases of the Respiratory Organs.*—On the part of the respiratory organs, the connection between surgical tuberculosis and latent pulmonary phthisis is a prognostic factor of considerable moment. This is emphasized in cases of anal fistula.

Careful consideration must be given to the functional capacity of the opposite side in all operations upon the dual organs of the body—kidneys, testicles, ovaries, special sense organs, etc.; *e.g.*, in the operation of thoracotomy the functional coefficient of the opposite half of the chest is of prime importance.

Pulmonary embolism is an untoward accident occasionally complicating even aseptic operations. It is particularly prone to follow upon pelvic operations, especially where there has been much bruising of the tissues or slight septic invasion. Either of these two factors gives occasion to extensive coagulation in the pelvic veins and sinuses, and the coagula may become detached and be swept along to the lungs in overwhelming masses. They may even prove instantly fatal by blocking the right side of the heart.

Fat embolism is most conspicuously met with in the lungs, and gives rise to a distressing dyspnœa, lasting until such time as the heart may be able to force through the pulmonary capillaries the embolic droplets of oil. Fat embolism

has been noted with relatively great frequency in the insane, as a sequel to multiple contusions. It is not uncommon after extensive omental resections, but is most frequently noted after fractures of the shaft of the long bones. The outcome of a case of fat embolism is chiefly dependent upon two factors—the amount of fat within the blood-vessels and the reserve power of the heart.

(*d*) *Nervous Diseases.*—The degenerative diseases of the nervous system have not much effect upon surgical prognosis, except those involving the anterior ganglia of the spinal nerve roots and transverse lesions of the spinal cord. In these the nutrition of the parts deriving their innervation from cord segments at or below the seat of the lesion is more or less compromised. Anterior poliomyelitis, locomotor ataxia, disseminated spinal sclerosis, and paralysis agitans do not of themselves seriously impair the outcome of such surgical treatment as may be demanded.

Insanity, idiocy, and epilepsy do not greatly affect the prognosis of surgical treatment. As Mayo * tells us, the insane are entitled to just the same surgical treatment as the sane—no more, no less. The relative frequency of fat embolism in the insane has been noted above.

With regard to the reciprocal effect of surgical operations upon the cerebral or mental lesion, there is little that can be stated definitely. Some few cases have been brought forward by Italian surgeons to vindicate a traumatic origin for insanity, alleging that the neurosis was caused by a variety of surgical operations; but when we examine the cases reported, we find that all the patients were strongly predisposed neuropathic subjects, with unfavorable environment, and that the operations either were upon the genital organs, or else occasioned some bodily disfigurement which gave the patient great annoyance. Yet just these conditions might evoke the neurosis in others, and the cautious surgeon might do well to bear this possibility in mind if called upon to operate in certain predisposed individuals. The effects of surgical operation upon the established neurosis are extremely problematical, and, while cures have followed surgical relief of chronic peripheral irritants (pelvic and abdominal tumors, operations upon the male and female genitalia), yet, as Mayo advises, the criterion is not the probable effect upon the mental, but rather that upon the general somatic condition.

In considering surgical prognosis with reference to the affections of the nervous system, we must take account of the subject of shock. We have already discussed this matter with reference to the administration of the anæsthetic; we must now add a word with regard to the advisability of operative intervention *per se*. As we have already seen, shock manifests itself as a paresis of the vasomotor system. The chief danger is sinking of the blood pressure, owing, as we have said, to acute hemorrhage, to cardiac failure, or to vasomotor paresis. The extremely bad prognosis in operations upon patients in shock is hardly due

* Mayo: Med. Record, 1901, vol. 60, p. 173.

to the additional hemorrhage, because with modern methods of hæmostasis this can, with sufficient care, be kept within very small limits; nor is it due to cardiac weakness *strictiori sensu*, because we may be dealing with a heart that is without valvular lesion, without toxic myocardial degeneration, and that may not have been fatigued by over-exactions of work. Aside, then, from the question of supporting the anæsthetic, which we have already discussed elsewhere, inasmuch as we can put aside both additional hemorrhage and cardiac insufficiency, in the stricter sense of that term, it follows that the problem of prognosis in conditions of shock hinges upon the ability of the vasomotor system to withstand the additional irritation incident strictly to the further surgical manipulations, without having the vasomotor paresis deepen to complete vasomotor paralysis, which is death. For this reason full ether anæsthesia will be safer than semi-anæsthesia, especially than semi-anæsthesia with chloroform. It is the common consensus of railroad surgeons, who see the most cases of acute shock, that if hemorrhage has been arrested, and further irritation of the sensorium has been removed by keeping the patient at rest (with morphine if necessary) and protected from further loss of bodily heat, the prognosis is much improved by waiting for at least a partial subsidence of shock before undertaking any considerable surgical procedures.

Neurasthenia is likewise a subject which demands reciprocal consideration. Of this question the first phase is the effect of neurasthenia upon the outcome of surgical conditions; and the answer is that the effect is little or none, excepting in those types of neurasthenia which interfere with the general bodily nutrition.

To the second phase of this question, to wit, the effect of surgery upon the neurasthenia, it is very difficult to give a satisfactory answer. If the surgical operation removes an irritant antedating in its history the onset of the neurasthenia, and one which may be deemed to be the cause of the neurasthenic condition (*e.g.*, chronic appendicitis, uterine or perineal lacerations, chronic prostatitis), a good effect upon the neurasthenia may be expected from the operation. If the surgical troubles do not stand in a causal relation to the neurasthenia, the effect of operation *per se* may be to deepen, rather than to relieve, the neurasthenia; yet it should be remembered that by skilful suggestion on the part of the physician he may array the powerful impression of a surgical operation upon the imagination of a suggestible patient, among the forces working for, rather than against, recovery from the neurasthenia. It is in so far as they induce in the subject oftentimes a quasi-physiological condition of neurasthenia, that menstruation, pregnancy, and the menopause may affect surgical prognosis. Too frequently repeated pregnancy and too prolonged suckling act in the same way.

Emotional conditions are not without their bearing, as is seen in the comparative mortality of the wounded among the victors and among the vanquished on the battlefield.

The effect of over-fatigue, exhaustion, exposure, and hunger is obviously unfavorable.

(*e*) *Diseases of the Alimentary Tract.*—A condition of the alimentary tract most unfavorable to surgical prognosis is one where, owing to either motor or secretory disturbances, that condition is developed which has been crudely called "stercoræmia," inasmuch as it magnifies the inevitable post-operative discomforts and disturbances to a degree which may become very serious; vomiting, headache, meteorism, obstinate constipation, and colic are bugbears of the abdominal surgeon, and have a direct connection with intestinal putrefaction and absorption. On this account a certain degree of gravity attends all operations done without preliminary evacuation of the alimentary canal; and this explains in considerable measure the high mortality from strangulated hernia and other forms of mechanical obstruction of the intestines. Mere obstipation —that is, the loading of the bowel with large masses of hardened fæces—is of less unfavorable moment than the presence within the intestines of a much less considerable amount of fecal material in a softer and more fluid condition, permitting the more ready absorption of toxins generated by intestinal putrefaction.

When to the products of the general putrefaction of the intestinal contents are added the specific toxins of disease, a condition which is conspicuously present in typhoid fever, the gravity of the prognosis, when operative intervention becomes imperative, is undoubtedly greatly increased. Yet surgery has recently been invoked with considerable frequency for the repair of intestinal perforations in typhoid fever, and with a success which has been on the whole gratifying. Gesselewitsch and Wanach * collected reports of 63 operations with 11 recoveries. The most recent reports show a slightly better percentage.

In considering lesions of the liver in their relation to surgical prognosis, it is important to bear in mind the difficulty attending upon a proper hæmostasis in cases of cholæmia, on account of the deficient coagulability of the blood. So considerable is the danger of hemorrhage from this cause that, where it is possible, operations not of the greatest urgency should be postponed until after the subsidence of the cholæmia, or until the exhibition of calcium chloride or other suitable drugs shall have demonstrated a fairly satisfactory rapidity of coagulation upon suitable tests.

In obstructions of the common duct the likelihood of the supervention of acute hemorrhagic pancreatitis and fat necrosis is an important prognostic element. In estimating the prognosis in any disease of the chylopoietic system, the determination of the stage, in what may be called the "biliary sequence," in which the patient under consideration may find himself, is an important prognostic moment; that is, his chances, if he be suffering from cholecystitis, of having this develop a cholelithiasis, and this in turn an obstructive cholangitis,

* Quoted in von Bergmann and Bull's "System of Practical Surgery," vol. iv., p. 319, New York, 1904.

whence perihepatitis, pancreatitis, and, eventually, pancreatic diabetes. In questions of this kind the family history acquires great prognostic importance. In a family in which there may have been several diabetics, the earlier stages in the "biliary sequence" have a less favorable prognosis than when occurring in families not so predisposed.

(*f*) *Diseases of the Ductless Glands.*—The danger in operating upon those who are the subjects of Graves' disease seems to come principally from the side of the heart. Aside from thyroidectomy and other operations directed specifically toward the cure of the disease itself, the special prognosis of which operations will be discussed elsewhere in this work, the warm extremities of sufferers from Graves' disease, due as they are to wide vascular dilatation and a rapidly flowing blood stream, are an indication of conditions favorable for the repair of the tissues, if only the heart be able to sustain the depression due to anæsthesia and the shock incident to operation.

In myxœdema, on the other hand, while the danger from the side of the heart is less, the condition of the tissues is less favorable for the repair of trauma and for resistance to microbial invasion.

(*g*) *Diseases of the Blood.*—In considering the bearing of the condition of the blood upon surgical prognosis, we touch upon a theme that will be handled more fully elsewhere in this work under the head of surgical hæmatology (see page 555). The simple question of hæmoglobin percentage has an important bearing upon prognosis, especially with regard to cases which it is proposed to submit to operation. For weighty observations on this point we are specially indebted to von Mikulicz. He has studied in patients, in whom conditions were otherwise favorable, but who had suffered considerable loss of blood by acute hemorrhage, the length of time necessary for the restoration of the blood mass, and he finds a period of from two to five days necessary for its restoration where less than one per cent of the blood in the body has been lost; while, with a loss of between three and four per cent of the blood in the body, from fourteen to thirty days are required to make good the loss of blood. These are more or less fixed data which will govern the prognosis for operations upon otherwise normal healthy individuals. A pre-existing anæmia, indicating a deficient hæmatopoietic function, will add greatly to the periods of time necessary for blood regeneration; and, according to von Mikulicz, a hæmoglobin percentage below 30 is a positive counter-indication to operation, until medical treatment shall have enriched the blood to at least this critical minimum.

Aside from the questions of blood mass and hæmoglobin percentage, the new science of hæmatocytology has brought for our use many facts bearing directly upon general surgical prognosis. They can be but briefly alluded to here.

The red-cell count is of great prognostic importance, and a diminution to anything below 3,500,000 must be considered to compromise the prognosis most seriously in all cases not due to acute hemorrhage.

Leaving for proper treatment elsewhere the interpretation of differential blood counts, we may mention here, because of its direct bearing upon our subject, the salient points in which leucocytosis indicates the prognosis in surgical cases, and especially in pyogenic processes.

1. The degree of leucocytosis is independent of the amount of pus.

2. An increasing leucocytosis points to a spreading pyogenic process.

3. The absence of leucocytosis in the undoubted presence of pus indicates:

(α) That the pus has become sterile, owing to the death of the invading bacteria.

(β) That the pus has been thoroughly walled off by granulation tissue.

(γ) That the case is of the "fulminating type," and of such virulent onset that a prophylactic chemotaxis has not established itself.

The slow coagulation of the blood in cholæmia and the prognostic importance of hereditary hæmophilia have already been alluded to.

(*h*) *Acute Infectious Diseases.*—The presence of acute infectious disease will undoubtedly counter-indicate any but the most urgent surgery. Particular stress should be laid upon *measles*, on account of the increased danger in anæsthesia, owing to the accompanying bronchitis; *scarlet fever*, on account of its intimate relation with erysipelas; *smallpox* and even *chickenpox*, on account of the pustular character of the eruption; and *diphtheria*, on account of the extreme probability of local wound infection. In addition to these, *pertussis*, besides the danger to the lungs from the anæsthetic, is particularly dangerous in abdominal surgery, on account of the impossibility of securing rest to the abdominal walls. *Grippe*, furthermore, is a great foe to the surgeon, as it renders its subjects particularly vulnerable to the attack of pyogenic germs. Some striking examples of this have been observed and reported by Bennett.*

(*i*) *Constitutional Diseases.*—The diseases of metabolism, so-called constitutional diseases, which particularly concern the surgeon, are four—rickets, syphilis, gout, and diabetes. The relation of *rickets* to general prognosis is simply that of malnutrition in general, the prognosis of the specific bone lesions of rickets being properly a part of special prognosis. When bodily nutrition is otherwise good, wounds, so far as my observation goes, heal as well in rickety subjects as in others.

Syphilis, if recognized and if met by suitable internal medication, need not deter the surgeon from such operative measures as may be deemed advisable; if unrecognized and untreated, it may hinder seriously the powers of repair, and operation wounds may prove to be a *locus minoris resistentiæ*, upon which a gummatous process may become engrafted with disastrous results. The consideration of the outcome of the surgical treatment of specific syphilitic lesions (gummata, etc.) lies in the field of special rather than in that of general prognosis.

* W. H. Bennett: "Brief Notes of Some Cases of Pyæmia and Suppuration, Apparently Due to the Prevailing Epidemic of Influenza," Lancet, Lond., 1890, i., p. 200.

Gout has its chief surgical interest in two points: first, the vulnerability of its subjects in presence of microbial invasion; and, secondly, the probable existence of gouty nephritis, not distinguishable in its symptomatology nor in its prognostic importance from other forms of chronic nephritis.

Of very special interest to the surgeon, on the other hand, is *diabetes*. The disastrous effects of surgical operations upon diabetics have led some to the absolute proscription of all operations in the presence of this disease. This extreme position we cannot, however, maintain, and it behooves us to consider this question, as we have several others that we have met, in its reciprocal relations; that is, first, What is the effect of diabetes upon surgical procedures and conditions? and, secondly, What is the effect of surgical procedures upon the diabetes?

The first question ranges itself in a class strictly parallel with other chronic dyscrasiæ, such as gout, syphilis, or chronic alcoholism; and acute dyscrasiæ, such as scurvy and the toxæmias of acute disease, in that diabetes causes a very greatly increased vulnerability of the tissues and renders them particularly susceptible to the invasion and development of pyogenic germs. Thus furuncles, carbuncles, and other phlegmonous and gangrenous processes will follow upon slight infection. Operations especially dangerous in diabetics are those, like circumcision for diabetic balano-posthitis, where rigid asepsis is difficult or impossible. The low resistance of the tissues in diabetics is, furthermore, influenced by the chronic endarteritis which this disease induces.

The other phase of this question, viz., the effect of surgical procedures upon the diabetic process, bears a still more forbidding aspect, and diabetic coma looms as a dreadful spectre before the surgeon called upon to operate upon diabetic patients, and the difficulty is to find a proper criterion by which to judge of those in whom coma is likely to develop. There is no one factor that will serve to guide us, and our chief dependence must be in the summation of the different evidences of grave diabetes.

Of these, the age of the patient has considerable weight; the more advanced age being in this case more favorable as regards prognosis, inasmuch as "diabetes gravior" appears more frequently in the young, and "diabetes mitior" more frequently in the aged. The absence of the patellar reflex, though it is not conclusive evidence of a grave diabetic condition, yet must be considered an unfavorable symptom. The coexistence of albuminuria and glycosuria, and more particularly the substitution of albuminuria for a pre-existing glycosuria, are of great importance. Cachexia and emaciation do not seem to have as much weight as might be supposed, apparently well-nourished individuals of vigorous appearance succumbing as rapidly as others. Sufficient recorded observations are not available to enable us to judge as to the prognostic value of heredity in diabetes—that is, whether a diabetic patient, a member of a diabetic family, is more liable to post-operative accidents than is, *cæteris paribus*, a diabetic patient not so related; nor do recorded observations throw any light as to the liability

to post-operative accidents of diabetics whose diabetes comes as the concluding member of the "biliary sequence," as compared with those whose diabetes has had no antecedent cholelithiasis, biliary stasis, and chronic pancreatitis. Both of these points, however, merit consideration.

When we consider the characteristic urinary excreta of diabetics, we can dispose of the polyuria and of the amount of sugar as not in themselves conclusive, though their behavior under appropriate dietary and medicinal treatment is of some prognostic weight.

The other characteristic excreta are acetone, aceto-acetic (diacetic) acid, and beta-oxybutyric acid. It seems, from chemical grounds, reasonable to suppose that beta-oxybutyric acid is the first of these to be formed in cases of beginning diabetic "acidosis," and that, when the disturbance of metabolism is not too severe, this is further oxidized to aceto-acetic acid, and then to acetone and carbonic acid. With further disturbance of metabolism, the oxidation does not proceed beyond the stage of diacetic acid, which accordingly is found in the urine; while, with still further metabolic disturbance, beta-oxybutyric acid appears in that secretion unchanged. Acetone, diacetic acid, and beta-oxybutyric acid in the urine are of successively important prognostic significance when it is proposed to attempt surgery upon diabetic subjects.

The appearance of aceto-acetic acid and of beta-oxybutyric acid in the urine (also other acids, æthylidene lactic acid, alpha-oxypropionic acid, and transitory fatty acids found in the urine of diabetics) is the sign of beginning acidosis, and manifests itself by an increase of the excretion of ammonium in the urine, in that the acids which appear in the blood are combined with ammonium.* A quantitative estimate of ammonium in the urine might, therefore, be of prognostic value. The method recently suggested by Folin † is not too elaborate for clinical use in an important case.

The simple examination of cover-glass preparations of the blood for free fat droplets ‡ would also suggest itself as a reasonable precaution. Great increase in the normal fat content of the blood (0.75–0.85 per cent) is characteristic of severe diabetes, in which amounts varying from 1.276 per cent to 11.7 per cent have been encountered. In such cases fat embolism may be found post mortem, plugging the vessels of various organs, notably the brain, the lungs, and the kidneys; and this fat embolism may account for many of the untoward post-operative accidents in diabetes. The preliminary dyspnœa which ushers in diabetic coma is probably due to this cause.

To sum up, a careful surgeon, before undertaking operation upon a diabetic subject, would do well to make the following preliminary investigations: 1, The

* Leube: "Medical Diagnosis," New York, 1904, p. 828.

† O. Folin: Zeitschr. f. physiol. Chem., vol. xxxii., p. 575; found also in Simon's "Clinical Diagnosis," Phila., 1902.

‡ Simon: *Op. cit.*, p. 56.

age of the patient; 2, the heredity; 3, the history of previous biliary trouble; 4, the condition of the patellar reflex; 5, an examination of the blood for excess of free fat; 6, the presence of albuminuria; 7, the behavior of the polyuria and the glycosuria under appropriate treatment; 8, the determination of acetone, diacetic acid, and beta-oxybutyric acid; 9, if any one of these three substances is found in the urine, a determination of the total ammonium excretion.

(*k*) *Concomitant Pyogenic Disease.*—Another matter calling for consideration before undertaking surgical operations is the possibility of the existence of a latent and concealed nidus of pyogenic infection outside of the field of operation, as such a smouldering process may be greatly quickened by operative manipulations even at some distance from such a focus, and may induce septicæmia or pyæmia, in spite of a faultless operative technique. Chronic encysted foci of osteomyelitis, and chronic but quiescent cholangitis, are particularly liable to take on such untoward activity; but perhaps the most conspicuous case of all is when virulent pelvic inflammation develops after a simple uterine curetting, done in the presence of a quiescent pyosalpinx.

3. Special Disease Conditions Bearing upon General Prognosis.—Inasmuch as it is well recognized that the preliminary stages of anæsthesia—what with the struggling of the stage of excitement, and the spastic closure of the lips and glottis, and the vomiting—are often attended by a temporary rise in the blood pressure, it is well to notice in this connection certain conditions already alluded to in considering the prognostic importance of parturition, where this rise of blood pressure might occasion rupture or harmful distention of the blood-vessels. The principal conditions in this category are aneurisms, varices, and some intracranial affections. In the operations of cataract extraction and even simple iridectomy, the possibility of collapse of the ocular globe and escape of the vitreous humor through the wound must be borne in mind. Struggling of the patient during preliminary anæsthesia may greatly affect the prognosis in the case of certain thin-walled abscesses and cysts, which may burst and carry their infectious contents into the great serous cavities.

V. Environment.

Under the head of environment we must consider not only the literal material objects surrounding the patient, but we must use the term in its wider sense, so as to cover the relation established between the individual and external objects, so that we may, as already suggested, include in our study of environment his occupation, his food, the climatic conditions under which he lives, his ability to create about himself hygienic conditions, and his disposition through habit or training to observe the laws of hygienic living.

Yet first, in the more restricted sense of the word "environment," we should consider such questions as the prognosis in emergency or field surgery as com-

pared with the prognosis under hospital surroundings; evidently many cases of good prognosis under hospital conditions would have a less favorable prognosis on the battle-field or in the railroad wreck. But, on the other hand, even a well-appointed hospital is not without its own proper disadvantages; for instance, a succession of cases of tetanus or of erysipelas occurring in a certain operating-room or hospital ward may most unfavorably affect the prognosis in a whole group of cases. Other things being equal, so far as the material surroundings of the patient are concerned, the best prognosis should obtain in the private homes of reasonably well-to-do people.

Under this head of environment belongs the consideration of the social and economic status of the patient, his ability to secure good food, sufficient protection, proper attention to his wants, freedom from worry and excitement—all of which may cast the balance favorably or unfavorably as regards recovery, and should, in not a few instances, determine for us the therapeutic measures to be adopted. Some social circumstances justify risks which other social circumstances would not justify. To a typesetter or a pianist the loss of a finger joint would mean much more than to a laborer; and for a poor man with a large family, the amputation of a leg, with prompt recovery, might be a far less serious disaster than conservation of the limb after months of hospitalization and invalidism. An artificial anus or a permanent gastric or urinary fistula are horrible afflictions where cleanliness cannot be counted upon, yet they may afford months or years of comparatively comfortable living to those who can be properly waited upon or who understand and habitually practise a rational personal hygiene. In female patients of means and leisure, pelvic operations may be indefinitely postponed, when rest and luxury are available to mitigate surgical conditions which the harder life of less prosperous patients would make unbearable.

The personal habits of the patient bear most decidedly on prognosis. Sloth, gormandizing, sexual excesses and perversions, inordinate use of tobacco, or other stimulants or narcotics are prognostic factors of great weight. Chronic alcoholism is perhaps the one factor most generally inimical to the recovery of surgical patients in hospital practice, on account of the depraved nutrition of the tissues and their consequent low powers of resistance; on account of the diseased liver, kidneys, and blood-vessels of chronic inebriates; on account of the over-stimulated and unresponsive heart; and, lastly, on account of the liability to delirium tremens. A reliable criterion by which to gauge the likelihood of the supervention of delirium tremens is nearly as difficult to establish as is a criterion for estimating the probability of diabetic coma, yet I have endeavored to assemble below the points which would seem to me to indicate special liability to this accident. They are:

1. The age of the patient, or, rather, the length of time that he has indulged the drinking habit.

2. The occupation of the patient. This is parallel to the "moral risk" of the life-insurance actuaries, as it gauges more or less accurately the drinking habits of the patient. Proscribed occupations are liquor-dealers, wholesale or retail; drummers for such; bartenders and other employees in breweries, distilleries, and saloons; hotel-keepers, etc.

3. The acknowledged drinking habits of the patient. Habitual drinkers who are never "drunk" are worse subjects than are periodical drunkards, save when the latter present themselves for surgical treatment at the close of a severe spree.

4. Loss of the knee-jerk is observed in the quiet stage of alcoholism. It indicates a considerable degree of toxæmia and is of unfavorable prognostic significance. An exaggerated knee-jerk is seen in the stage of excitement, and, if accompanied by alcoholic tremor, it is still more unfavorable than is loss of the knee-jerk.

5. The well-known tremor of the hands is most unfavorable.

6. Alcoholic gastritis indicated by morning vomiting, anorexia, and inability to retain or digest food.

7. Starvation and exposure.

8. The combination of alcoholism with any demonstrable organic lesion of the liver, lungs, or the kidneys; or any constitutional disease, such as anæmia, gout, or diabetes; or any acute infectious disease, such as malaria or influenza.

A final factor to be considered is climate. A tropical climate is depressing to those not habituated to it and to those who have not learned or will not practise a reasonable tropical hygiene. In such subjects surgical operations have an additional element of peril, which can be greatly reduced by removal for operation to the temperate zones. The prognosis of many surgical diseases of the upper respiratory passages and of the thorax, and of many surgical affections of the urinary organs, is most favorably influenced by a dry and equable climate. The effect of sunshine and open-air life is no less marked upon surgical tuberculosis than it is upon those tuberculous affections of the lungs and other internal organs which come more especially under the care of the physician.

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